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ABSTRACT

Presented is a comprehensive survey of the literature on the relationships between disease and solid wastes. Diseases are grouped on the basis of waste type or disease vector, such as chemical waste, human fecal waste, animal fecal waste, rodent-borne disease, mosquito-borne disease and miscellaneous communicable disease. The following format is used in postulating the solid waste/disease relationships: first, a general statement on the disease being considered; second, a postulation on the disease association with wastes; third, supporting evidence found in the literature; fourth, discussion of the evidence; fifth, conclusions and possible projections relating to disease, waste association; sixth, recommendation for research and other activities. Appendices include an extensive list with the following information: solid waste sources, resulting wastes and their composition, and means of treatment. Included is a list of descriptors and a bibliography with 755 entries. (PR)

SOLID WASTE/DISEASE
RELATIONSHIPS

a literature survey

U.S. DEPARTMENT OF HEALTH, EDUCATION, and WELFARE
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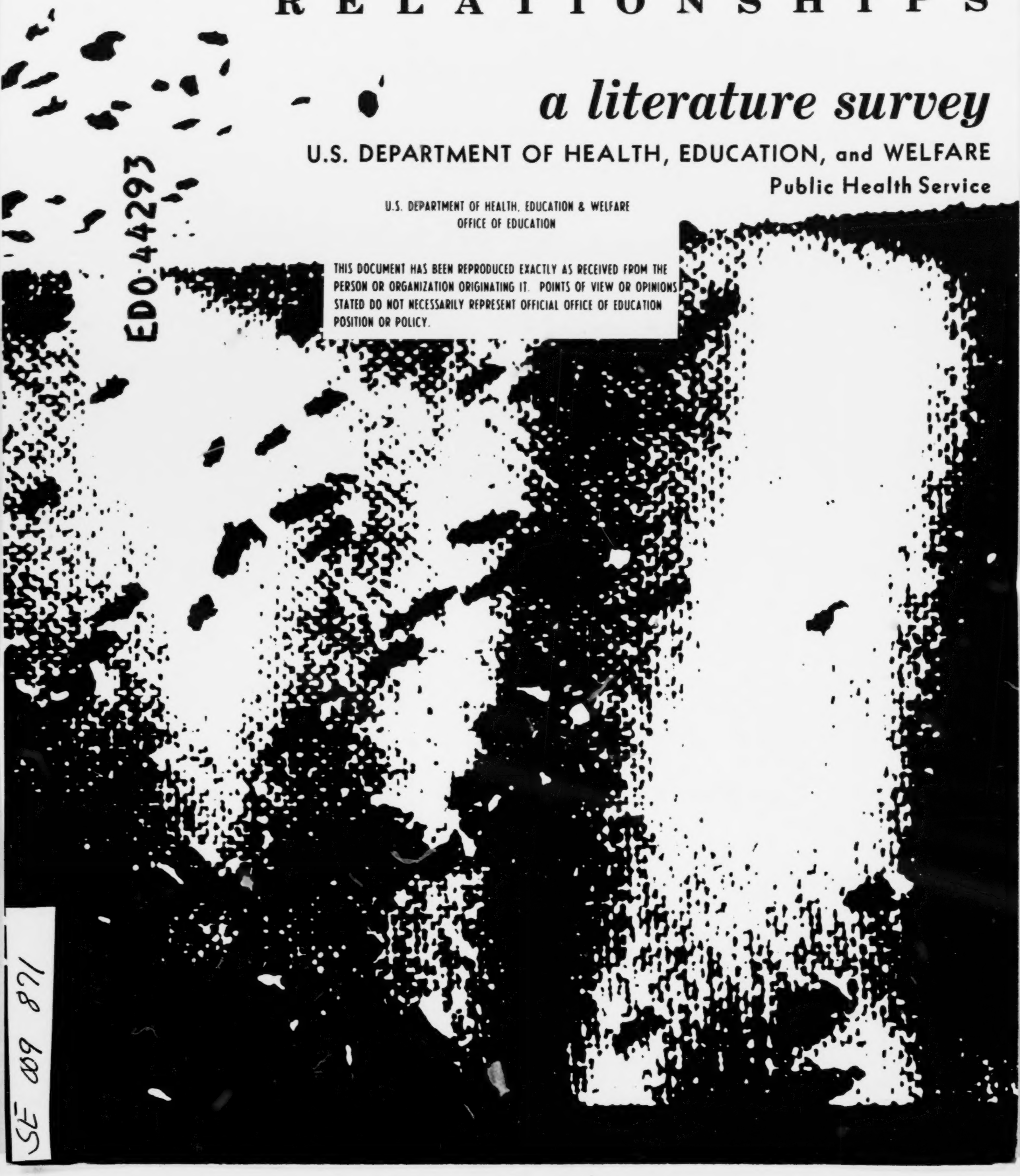

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SOLID WASTE / DISEASE RELATIONSHIPS

a literature survey

This report (SW-1c) was written for the Solid Wastes Program
by **THRIFT G. HANKS**, M.D.
Life Systems Division, Aerojet–General Corporation
under Contract No. Ph 86-66-151

U. S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE
Public Health Service
BUREAU OF DISEASE PREVENTION AND ENVIRONMENTAL CONTROL
NATIONAL CENTER FOR URBAN AND INDUSTRIAL HEALTH
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CINCINNATI
1967

The ENVIRONMENTAL HEALTH SERIES reports the results of scientific and engineering studies of man's environment. The reports provide information on research activities conducted and supported by Centers in the Public Health Service's Bureau of Disease Prevention and Environmental Control. The subject of each report is indicated by these letters:

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foreword

THE SECRETARY OF HEALTH, EDUCATION, AND WELFARE, John W. Gardner, has said that maintaining the quality of the human environment "is potentially the most important technological challenge of our age." This challenge will go unmet as long as efforts to dispose of solid wastes continue to cause extensive environmental pollution, place an incredible economic burden on the people of the United States, and contribute to the spread of a host of human illnesses.

Any one of these reasons would be sufficient to dictate a concerted national effort to improve solid waste disposal practices. But the fact that the health of the American people is seriously and needlessly threatened makes imperative the requirement that the solid waste problem be brought under lasting control.

It is my hope that this report, which attempts to summarize the current scientific knowledge of the health implications of solid waste disposal, will prove to be a landmark on the road toward safe and sanitary solid waste management. I hope that it will stimulate the additional research needed to close the gaps in present knowledge of solid waste disease relationships and that it will motivate scientists and others concerned with protection of the public health to turn their attentions to this vitally important health problem.

—RICHARD A. PRINDLE
Assistant Surgeon General
Director, BUREAU OF DISEASE PREVENTION
AND ENVIRONMENTAL CONTROL

preface

THIS IS A REPORT of a comprehensive literature survey of the public health aspects or disease relationships of solid wastes.

The study was conducted by the Life Systems Division of Aerojet–General Corporation, Azusa, California, under a contract with the Solid Wastes Program, United States Public Health Service. The study was directed by Thrift G. Hanks, M.D., who prepared the body of the report. Although there is a paucity of past work on the etiologic factors of solid waste, an attempt has been made to cover the field comprehensively enough to meet the needs of public health practitioners.

No single treatise in the past has attempted to correlate the available information as to various diseases directly or indirectly related to solid wastes. Such a work is obviously desirable because of the complexity of the solid waste/public health interface.

The facts brought together in this volume are widely scattered in literature and many of them were difficult to obtain. Although a logical plan was followed in deciding what to include in the report, many of its postulations and conclusions admittedly are difficult to uphold by documentation. There is no guarantee that all pertinent literature was retrieved, but there is a fair guarantee that documents representative of the total pertinent data were reviewed so as to give a reasonably valid statement of known relationships and of suspected or possible relationships as well as of lack of relationships. As a result, this document is an excellent and useful literature review.

The authors have not limited the text strictly to solid waste, but have drawn upon data from other etiologic modes in order to arrive at possible connections of solid waste and its by-products to disease transmission. Their inability to present a "proof positive" solid waste/disease relationship stems from the fact that our present state of knowledge is not sufficiently standardized to make possible the preparation of a concise account of the subject. The problem of identifying direct health effects is not unique for solid wastes, but occurs throughout the field of environmental health. However, the postulations made and the evidence cited in support provide potential means for interpretation of waste/disease relationships via different pathways and also enable the health worker to identify areas where more extensive research in the disease aspects of solid wastes is needed, particularly from the viewpoints of epidemiology, etiology, pathology, symptomatology, and prevention.

Mr. Ralph J. Black, Assistant Chief of the Solid Wastes Program, served as the project officer of this study.

—LEO WEAVER, *Chief*
Solid Wastes Program
NATIONAL CENTER FOR URBAN AND INDUSTRIAL HEALTH

THIS REPORT has been submitted to the Solid Wastes Program of the United States Public Health Service in partial fulfillment of CONTRACT NO. PH 86-66-151 which was performed during the period 15 June to 15 December, 1966. The report proper is contained in this volume, Volume I.

A list of references as prepared by the contractors follows immediately after the body of the report. Volume I also contains three appendixes. Appendix A is entitled *Research*, Appendix B is called *Solid Wastes Sources and Constituents*, and Appendix C is designated *Descriptor Glossary*. The annotated bibliography in the separate, unpublished volumes (Volumes II through V) has been microfilmed by University Microfilm Company, Ann Arbor, Michigan, who will be sole distributors.

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INTRODUCTION

IT IS FORTUNATE that persons concerned with public health have acted to control disease without waiting for the discovery of "ultimate scientific truth". Only now are some of the ecologic and epidemiologic details being established to justify essential public health measures instituted decades ago. Nevertheless "guilt by association" between solid waste and disease does not provide the kind of foundation needed to provide scientific data upon which public health directors, sanitary engineers, government officials, and the voting and participating public may act with confidence and a minimum of dispute in dealing with the enormity of the solid wastes problem in the United States.

The assigned purpose of this study, therefore, has been to review with reasonable thoroughness the scientific literature reporting any relationships between disease and the solid wastes generated by the activities of man. Thus the direction of this study has been epidemiologic in nature, and its methods have been based on the requirements of epidemiologic validation.

Theoretically, if all etiologic factors are present in a requisite quantitative and sequential manner, a necessary and sufficient climate for disease will exist, and the disease process may result. Any chain or cycle of events between agent and host, and within the pattern of the disease, must be unbroken. Each link must be firmly established by reproducible observations. Proven demonstration of solid waste/disease relationships is hampered by: the complexity of solid wastes components; the biologic and chemical changes that occur in wastes under widely varying conditions of storage, handling, treatment, and disposal; and the intricate pathways that may underline human exposure and disease. There is also the frequent need to isolate one among other possible etiologic modes, so that if there is a relationship the association may be hidden by more readily demonstrable, plausible, and significant etiologic mechanisms.

Despite these difficulties, the literature contains a number of statements implying or averring that significant connections between solid wastes and human disease exist. But the dearth of explicit relationships found at the start of the study led to a need to postulate the essential links, then to attempt to determine such connections as might be indicated by isolated published observations, and finally to the attempt to establish or exclude each linkage in an epidemiologically satisfactory manner. Some of the postulations are diagrammed in the text of this report. Each postulated solid waste/disease relationship is presented as follows: First, a general statement on the disease under consideration; second, a postulation on its association with wastes; third, the evidence found in the literature supporting this postulation; fourth, discussion of the evidence; fifth, conclusions relating to the disease/waste association and to possible projections of the observations; sixth, recommendations for research or other activities.

We do not apologize for some of the extensive explorations carried out in pursuit of solid waste/disease relationships. Essentially, each search was based on a published statement or opinion that a relationship could, or did, exist, and only by thorough examination could we test the validity of such a statement. At times the examination required an investigation of evidence pertaining to factors seemingly unrelated to solid wastes in order to infer chance or indirect contributions of solid wastes to the spectrum and incidence of disease.

It should not be surprising that so much opinion and so little data were discovered in this study. The complexity of the subject matter, the obscurity of disease pathways, and the absence of reliable methods contributed to the paucity of scientific information in this area. Tradition and teaching dating back half a century, however, have tended to fix certain beliefs for which valid demonstration has only recently been established. Yet despite any shaky foundations in older public health lore, we may well be grateful that our pioneers in public health acted on limited data and much intuition. We are a healthier nation for it. The lesson may help to support the initiation of action in our present problems concerned with the management of solid wastes before all demands for proof positive are met.

Acknowledgements

Robert E. Mitchell abstracted and summarized the material on waste sources and constituents, proposed and conducted the safety survey, and (with the assistance of Rex Farquhar, statistician) analyzed the survey data. Robert Geckler, PH.D., prepared the summary on research (Appendix A). Perry Ann Stith, M.S., researched a significant portion of the chemical waste/disease citations.

Special recognition is given to Myra Grenier, Aerojet Corporation Librarian, and her staff, particularly Shirley Stephenson, for their splendid support.

Among the library sources, particular thanks go to the Linda Hall Library, Kansas City, Missouri. Local sources provided invaluable help. Elizabeth Acker, head of the reference department of the Los Angeles Medical Society Library, gave gracious support. The libraries of the California Institute of Technology, Pasadena, University of California at Los Angeles, University of Southern California, and the City of Hope Hospital, Duarte, California, were used extensively.

—THRIFT G. HANKS

RATIONALE AND METHOD

A LITERATURE SEARCH involving solid waste and disease relationships imposes a choice of primary descriptors, in that the selection of the universe of published disease processes would overwhelm the searchers. It was therefore decided that the term *waste* and its synonyms and near-synonyms would be used as the primary search terms, while postulated or cited disease and other descriptors would serve as secondary search terms.

A sampling of literature indexes was made. This involved use of the initial terms arrived at by the description of the study, and the experience of the study personnel. An expansion of the initial descriptor list was then prepared from the terms found under the cross-indexing of the indexes. A cyclical search plan was set up and extended to include activities leading to a final report. The resultant survey plan is shown in Figure 1.

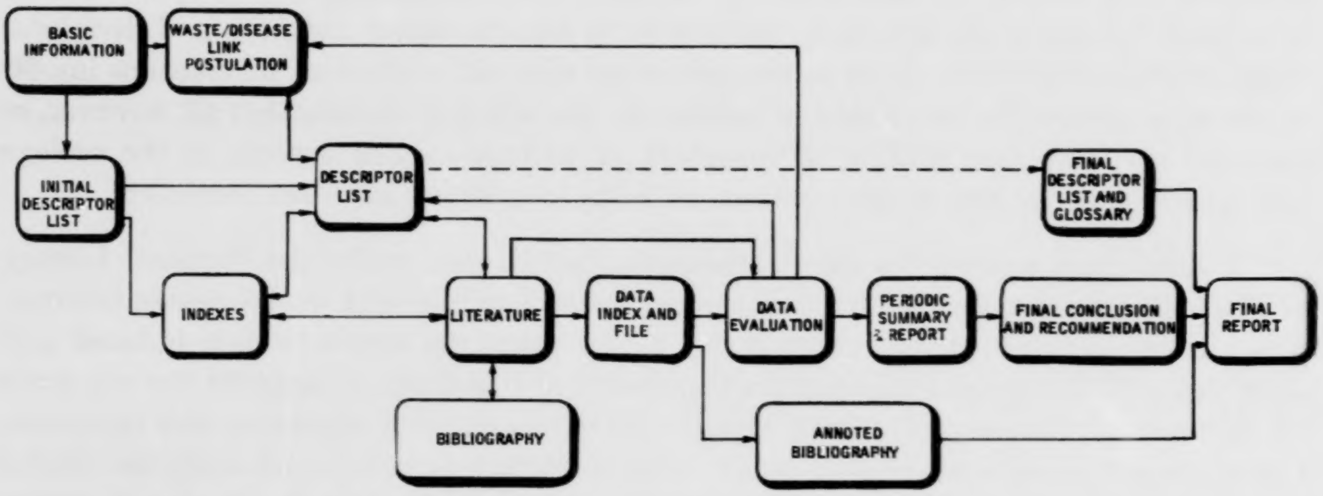
BASIC INFORMATION
WASTE/DISEASE LINK POSTULATION
INITIAL DESCRIPTOR LIST
DESCRIPTOR LIST
FINAL DESCRIPTOR LIST AND GLOSSARY
INDEXES
LITERATURE
DATA INDEX AND FILE
DATA EVALUATION
PERIODIC SUMMARY & REPORT
FINAL CONCLUSION AND RECOMMENDATION
FINAL REPORT
BIBLIOGRAPHY
ANNOTED BIBLIOGRAPHY

FIGURE 1. Survey plan.

In support of the search, postulated waste/disease relationships were diagrammed. A few of these diagrams are contained in the report (Figures 2 through 6).

It was evident that a statistically random sampling of the literature could not be relied on to discover a sufficiently inclusive selection of existing references to satisfy the intent of the search. Instead, a screening of titles was used for which the descriptors were chosen to match each link of the postulated connections between waste and disease. Since *a priori* construction of all links was not possible, some repetitive searching of indexes was necessary. However, the method tended to limit the number of titles. On the other hand, a sample title search, using a postulated disease alone as the descriptor, resulted in no selections from approximately 11,000 titles.

More than 200,000 titles were screened before tabulations were discontinued. It is estimated that approximately 350,000 titles in all were scanned for selection. When possible, initially selected titles were further screened by examination of the abstracts before final selection was made. The indexes used were *Index Medicus*, *Chemical Abstracts*, *Biological Abstracts, Applied Science & Technology, Engineering Index, International Abstracts of Biological Sciences, Industrial Arts Index, Psychological Abstracts, Annual Review of Psychology,* and *Readers Guide to Periodical Literature.*

From an original list of 1,727 selected titles, 1,236 were received, read, and abstracted. The remainder were either not available or were cancelled when circumstances directed. (Translation of Slavic literature was not available, for example.) The physical method used for abstracting was 'highlighting' by colored ink, with broad felt pens to mark material for typing. Although this method did not always provide smooth sequencing of quotations, it did permit a time saving of at least twenty-fold over written abstracing, and five- to ten-fold over dictation.

From the perused 1,236 articles, books, reports, proceedings, and other sources, 754 abstracts were chosen for reference and inclusion in the annotated bibliography. We believe that the pertinent literature retrievable by feasible search methods and descriptor usage has been identified, either in the referenced material or through its citations insofar as the basic purpose of the study is concerned. No pretense is made that all, however, or even the key, literature relating to important or perhaps crucial aspects of the ecology and epidemiology of the disease, vectors, or hosts in question has been identified.

As a check against the manual methods used in this study, the National Library of Medicine was asked to use its MEDLARS system to retrieve solid waste/disease information. It should be noted that through this source there was access to data indexed only after 1963. With the use of descriptors supplied from this study, a computer run was made on all available literature (477,000 titles). From this run, 195 references were recovered. Unfortunately, these references were not available in time to obtain and study the articles not duplicated in the manual study. Since only 27 references were duplicated, however, the relevancy of the remaining 168 is unknown and should be determined. Correlation of the references obtained by the two methods would appear of fundamental importance to any retrieval system to be proposed for solid waste information.

A third requirement dealing with hazards of sanitation workers is discussed in the section on safety in this report.

SUMMARY AND CONCLUSIONS

THE LITERATURE FAILS to supply data which would permit a quantitative estimate of any solid waste/disease relationship. The circumstantial and epidemiologic information presented does support a conclusion that, to some diseases, solid wastes bear a definite, if not well defined, etiologic relationship. The diseases so implicated are infectious in nature; no relationship can be substantiated for noncommunicable disease agents associated with solid wastes, not because of negating data, but because of lack of data. (An exception to this statement may exist in the instance of methemoglobinemia of infants in which nitrates of excretory origin may play a part.)

The communicable diseases most incriminated are those whose agents are found in fecal wastes — particularly human fecal wastes. Where these wastes are not disposed of in a sanitary manner, the morbidity and mortality rates from fecal-borne diseases in the population are high. Despite the fact that other factors are known to contribute to some reduction of these rates, the inescapable conclusion is *that the continued presence in the environment of the wastes themselves is the basic causative factor.* Therefore transmission — whether by direct contact, vector transfer, or indirect contact — is due to environmental contamination by these wastes.

Flies are carriers of many disease agents, however, and fly-control experiments indicate that they are significant transmitters of shigellosis (bacillary dysentery). The known ability of fly vectors to proliferate enormously in organic wastes, to contaminate themselves in fecal wastes, and then to contaminate man or his environment, incriminate the fly as a secondary hazard. The wastes from which the fly arises, or by which it is contaminated, thus constitute the primary hazard. In other words, any solid waste which promotes fly propagation will contribute to the incidence of a disease when the agent of that disease is available to the fly, and when other conditions of transmission (for example, the ability of the fly to transmit the agent, proximity of flies to hosts, dosage factors) are satisfied. Since these contributory conditions may vary significantly from place to place and from human population to human population, the definitive factor must be the domestic fly population, which in turn is largely regulated by the breeding opportunities afforded by numerous solid wastes.

The importance of solid wastes to mosquito-borne disease is far less clear. The relative contribution of solid waste-bred as opposed to that of other media-bred mosquitos has not been studied. The inference to be drawn from available information is that, under certain circumstances (see section on mosquito-borne disease), the presence of breeding places provided by solid wastes could contribute to dissemination of a disease agent in a human population, although to an unknown degree.

At this time, few human cases of rodent-borne zoonoses are being reported in the United States. No recent data were found bearing on the relative importance between solid wastes and other media in contributing to domestic rat populations, thus incidence of the reported zoonoses cannot be estimated in relation to solid wastes. Human plague experienced in recent years in this country seems to be totally of direct sylvatic origin. The importance of the domestic rat in the few reported cases of leptospirosis cannot be judged because of insufficient epidemiologic data. The direct origin of the leptospirae responsible for human infection were largely of bovine or other domestic animal origin. The further tracing of the organisms to rats was not accomplished (see rodent-borne disease section). Other solid waste/disease relationships must remain speculative pending far more definitive studies. Because of the low level of incidence of a number of diseases for which a relationship could be postulated, it would appear that there is little hope of obtaining data sufficient for analysis in this country, and that applicable studies would have to be made on foreign populations if a more valid comprehension of an association is to be made.

The fact, however, that much basic and many 'tag ends' of research attesting to specific relationships or their degree have not been carried out should not serve as an excuse for failure to act. It is known that insanitary disposal of fecal and food wastes is a cause of disease in this country. It may or may not be economically sound practice to investigate disease transmission under various waste and water treatment methods before undertaking large treatment projects. But in any case it does not appear to be sound practice to delay the installation of sanitary toilets and water supplies pending the outcome of such research.

So little is known about the kinds and degrees of contamination of the human environment by industrial and other chemical wastes, and the impact of trace amounts of such materials on human health, that their relationships to health remains essentially unexplored. This entire field is open to investigation; the implications for the research effort, of course, are as broad as the gap in knowledge.

One rather puzzling finding is the high accident frequency rates among sanitation workers (see section on safety). This warrants study of this population to explore reasons for these rates and to estimate the public health risk of solid wastes factors encountered in the study of this group, who have a more intimate exposure than the general public to disease agents contained in wastes.

GENERAL RECOMMENDATIONS

SOLID WASTES have been demonstrated conclusively to be associated with some diseases in the United States. Although the incidence of disease due to wastes is low in this country as a whole, it is demonstrably higher in certain groups — particularly those without general sanitation, including proper waste disposal means. In the chain leading disease from waste to human host, the major point of attack must be upon those wastes which contain disease agents or serve as sources of propagation for carriers of disease. Wastes must be so handled or treated that the pathogens they contain are destroyed — not merely reduced in numbers — and carriers of pathogens denied access to the wastes for breeding or sustenance. To the extent that known effective measures are not feasible at this time, research should be directed at the development of effective, yet practical, methods.

Since lack of data is extensive in regard to chemical wastes, two major paths of investigation are advised: first, delineation of the type and degree of contamination of the environment due to chemical wastes; and second, accelerated and long-range toxicologic studies on effects of chemical waste materials common to the environment in the concentrations found there. The knowledge needed is that of the effect of decades of exposure to trace amounts of the waste substances.

In addition, exposure and disease spectra of sanitation workers should be determined to provide comparative data, as well as to permit better protection of this group.

It appears that we need more information on the techniques and the organizational and administrative means for sanitary disposal of solid wastes in times of crisis or disaster. As a preliminary step, the available information should be assembled as a basis for determining what additional knowledge is required. Correction measures against disease cannot deal exclusively with a relatively limited aspect of an etiologic situation as broad as that associated with solid wastes. Educational and legal weapons are required. Considering the deficiencies of health education as a whole in America's school systems, it is not entirely appropriate to select the public and personal health aspects of solid wastes as the focus of expanded instruction on health. Yet from a system of education developed on this aspect of health, an inclusive health education program of value might arise. Certainly, some means developed for use in the schools is needed for breaking some children from the cultural morass of insanitary practices to which their birth and environment condemn them.

Education of industrial personnel, the general public, the medical profession, and government officials is an added requirement. Educational and motivational materials and techniques need to be developed for the accomplishment of these goals. Strict legal controls and their enforcement are mandatory. However, regulations must be based on reasonable standards. At the present level of knowledge, it is not possible to adopt standards directed at all aspects of environmental contamination, including sources of solid wastes. For example, research is needed to permit the development of standards on chemical, viral, and (to a lesser extent) other biotic contamination arising from solid wastes. In the interim, considering the tendency of contaminants to ignore present jurisdictional boundaries, the legal and governmental means necessary for the effective application of regulatory standards need to be developed.

Additional studies on environmental contamination by chemical wastes have already been suggested. Such studies probably could benefit from, or require, automated monitoring systems. Such systems could then be adapted to environmental control for the protection of health. Their development therefore deserves consideration for at least two basic reasons (economic, regulatory, and legal reasons also suggest themselves).

In this report, occasional reference is made to the hazard arising from compartmentalized approaches to the control of environmental pollution. In almost every action to be recommended for the prevention of solid waste-related disease, there is a parallel requirement apart, but not detachable, from the solid waste phase which relates to all environmental contamination. The same considerations apply in reverse to water- and air-pollution control measures. That is, corrective measures (or research directed at their development) cannot be considered separately from overall waste management problems. The obvious conclusion is that environmental health is not a subject for dissection. Specialists may be required for diagnosis, but the therapy must be integrated. The basic recommendation, therefore, is that of an integrated program of study, analysis, and action directed at applying the best talents of our society as a whole to the control of environmental pollution.

More detailed recommendations are contained in this report under the section on research and at the end of each section on disease.

DEFINITIONS

IT IS PERHAPS APPROPRIATE to explain the definition of solid waste given below. First, there is no consistent definition to be found in the literature or among workers in the field of waste studies. Second, a waste may undergo as many as three or four phases of solid/nonsolid existence from the time it is evolved until its 'final' disposal. For example, wastes that go into suspension or solution cannot be abruptly dismissed. Through evaporation, precipitation, adsorption, and other phenomena, such waste often reappears in solid form.

Today's so-called liquid wastes may become tomorrow's solid wastes if public health or other considerations dictate that liquid transportation of solids be discontinued. A similar future problem may apply to solid wastes, or the combustion products of wastes now discharged into the air.

To classify such wastes on an arbitrary or jurisdictional basis would at this time almost certainly result in the lack of timely consideration of research for handling them, should their present modes of disposal be prohibited. This in turn would result in delays and elevated costs.

The following definition is given with the above considerations in mind:

Solid Waste: Solid waste is that normally solid material arising from animal or human life and activities and discarded as waste, regardless of its mode of transportation, suspension, or modification. It therefore includes waste particulates suspended in air or water, and soluble waste solids contaminating water or soil.

The following definitions are similarly arbitrary:

Descriptors: Those terms which define, for purposes of information retrieval, the subject matter encompassed by this study.

Descriptor Glossary: The table of search terms found to be most helpful for information retrieval. It contains major descriptors, synonyms, or near-synonyms of these descriptors, and modifying terms which more specifically define the subject matter sought.

Disease Agent: Any organism or material capable of causing disease.

Waste Disposal: The final deposition of waste by man. This does not include its ultimate dissemination by forces other than man.

Waste Handling: The physical manipulation of waste involving human exposure to the waste's components.

Waste Sources: Those activities — domestic, municipal, industrial, or commercial — which generate waste.

Waste Treatment: The intentional processing of waste with the goal of changing its content as to quality or quantity, or both.

SOLID WASTE / DISEASE RELATIONSHIPS

A. general discussion

HYPOTHETICALLY, solid wastes can produce undesirable effects by biological, chemical, physical, mechanical, or psychological means. For example, human pathogens in human feces provide a biological threat, industrial wastes create chemical hazards, flammable materials involve a physical danger through fires or explosion, and broken glass and other sharp-edged wastes create mechanical hazards. These hazards, plus unsightliness, costs of waste disposal, special interest and jurisdictional disputes, threats to property, and other factors, provide a basis for possible psychological and behavioral disturbances.

To relate human disease, disability, annoyance, or other undesirable effects unequivocally to the production, disposal, or accumulation of solid wastes is not a simple procedure. It requires, in fact, that all of the steps in the pathway from solid waste to human affliction be validated by unequivocal measures. This validation depends on the

1) Discovery of harmful or potentially harmful agents in the waste, or,
2) Demonstration that such agents develop within, or in association with, the wastes.
3) Discovery of disease or other effects among the population which may reasonably be associated with these agents;
4) Demonstration of the pathways by which the effects are accomplished;
5) Demonstration of the absence of effects following interruption of these pathways by one means or another, or their absence in populations not similarly exposed.

Among the human population, conditions exist which satisfy requirement three above (that is, there are numerous cases of disease, injury, or behavior which reasonably could be suspected of arising from the *kinds* of hazards postulated). Perhaps the easiest to associate would be arguments arising out of jurisdictional or economic aspects of solid waste management.

The most difficult requirement to fulfill is the fourth. It will be helpful to consider the possible pathways.

Disease agents must find access to the body if they are to have an opportunity to cause an effect. The agent and the victim must have environmental association, therefore, either directly or indirectly. Direct contact could occur, for example, in the handling of a waste by the potential victim. Indirect contact could occur through transportation of the agent to the victim as, for example, through the means of a biological vector such as a fly, mosquito, or flea, by way of water supply contaminated by waste, or by airborne solid waste particles.

In detail, the pathways by which agents of disease could result in human exposure and possible disability can be highly complex, but a simplified diagram of the general modes of transmission can be presented (Figure 2). Except for their brief mention, the possible psychological effects of solid waste will not be discussed in this report.

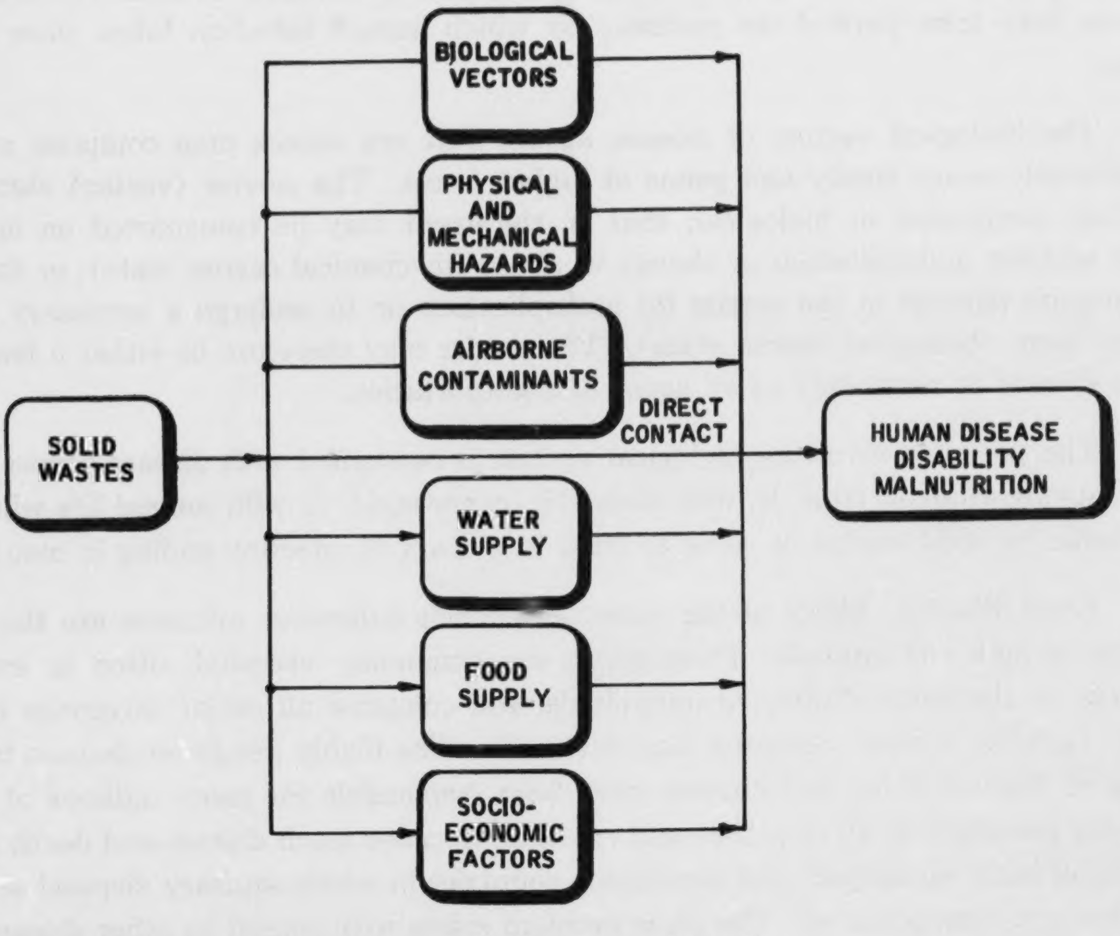
SOLID WASTES
BIOLOGICAL VECTORS
PHYSICAL AND MECHANICAL HAZARDS
AIRBORNE CONTAMINANTS
DIRECT CONTACT
WATER SUPPLY
FOOD SUPPLY
SOCIO-ECONOMIC FACTORS
HUMAN DISEASE DISABILITY MALNUTRITION

FIGURE 2. Solid waste/human disease pathways (postulated).

B. pathways

THE GENERAL CASES of the first six of the several pathways diagrammed above will be presented first. However, in treating individual diseases, overlapping of pathways permits no such separate categorizations.

Pathway 1: Biological Vectors

The term vector, as used here, refers to any living agent which transports, directly or indirectly, a disease agent. Later in this report it will have its more technical meaning — that is, an arthropod transmitter of disease. Vectors become important to the solid waste/disease relationship insofar as the waste is a cause of their multiplication and their proximity to man, or a source of their contamination by pathogens.

Biological disease agents affecting man have evolved and are still evolving. Some disease agents are adapted to lower animals and infect man only incidentally; that is, man is not a natural reservoir. In case of human disease from such sources, man can serve as a 'dead-end' for the agent, or he can transmit the agent among his own species or to other species. Other agents are totally or partially restricted to man. Lower animals, however, may form part of the pathway by which human infection takes place in such disease.

The biological vectors of disease agents that can attack man comprise members of essentially every family and genus of higher forms. The carrier (vector) status may be either mechanical or biological; that is, the agent may be transported on or in the vector without multiplication or change in phase (mechanical carrier state), or the agent may require passage in the carrier for multiplication or to undergo a necessary cyclical change there (biological carrier state). The carrier may therefore be either a true 'host' of the disease or serve only as an agent of transportation.

The pathway involving biological vectors is concerned with disease-carrier or host states among animals (that is, with domestic, commensal, or wild animal life which produce infective solid wastes or serve as links in a chain of infection ending in man).

Fecal Wastes. Many of the most devastating infectious ailments are the enteric diseases of man and animals. Their agents are commonly excreted, often in enormous numbers, in the feces of infected individuals, and comprise all major categories of pathogens: bacteria, viruses, protozoa, and helminths. The highly dangerous human bacterial agents of typhoid fever and cholera have been responsible for many millions of deaths. They are prevalent in all countries and continue to cause much disease and death in areas existing in both 'developed' and developing countries in which sanitary disposal of human feces has not been achieved. The same problem exists with regard to other disease forms found in feces, especially as a cause of death among infants and children.

Enteric diseases for which domestic animals are the natural reservoir may cause human disease through direct infection. By causing disease or death of the animals, these diseases can contribute to malnutrition and death in man. It is possible that there are hundreds of kinds of enteric animal disease agents. An unknown number are, or may become, transmissible to man either at present or, by evolution, in the future. Among these kinds are hundreds more of serological variants — infection with one of which may not provide immunization against other variants of the same species. For example, more than 1,000 serologically different salmonellae are recognized.[324, p.266] As world-wide trade and spread of transportation increases, the spread of species, subspecies, and strains of such organisms, at least among animals, will continue to mount.[324, p.171]

The discussion of specific diseases related to fecal wastes is presented elsewhere in this report.

Solid Wastes as Sources of Food and Harborage to Potential Carriers of Disease. The commensal rat and mouse are examples of evolutionary adaptations of a wild form to man's habitat. Their association, particularly that of the rat, with human refuse has been commented on throughout much of written history, as has their suspected role in human disease (see section on rodent-borne disease).

In addition, certain insects such as cockroaches, domestic flies, and domestic mosquitoes have been noted to share this relationship with human refuse and are suspected of spreading disease to man (see sections on fly-borne and mosquito-borne disease).

Pathway 2: Physical and Mechanical Hazards

Solid wastes often contain flammables and, in the course of biological or chemical decomposition, evolve explosive, poisonous, or asphyxiative gases. These gases traverse soil and can presumably create hazards to humans and their dwellings if these are proximal to the disposal, treatment, or other sites of processing of solid wastes. Solid wastes can also cause injury by mechanical means (see section on safety).

Pathway 3: Airborne Solids

Solid wastes are evolved in many combustion processes. A particularly heavy source of particulates is the burning of coal. Incineration of solid wastes creates solid matter which enters the air. It is also conceivable that pathogenic soil fungi, multiplying through the nutritive effect on soil of some solid waste constituents, can release spores to the air (see section on fungi).

Pathway 4: Direct Contact

It must suffice to surmise in the general case that many possibilities for direct contact of humans with biological or toxic agents in solid wastes must exist. In this regard, solid waste must be considered in two rather distinct contexts: first, as a collection and disposal problem involving sanitation workers, and second, as a problem of personal and household hygiene (at least insofar as an analysis of cause and effect relationships in disease is concerned). It will thus be necessary to consider both household and municipal pathways.

Possibilities for kinds of exposure are to be inferred from the sources and constituents of solid wastes. Exposure itself depends on proximity; hence occupations causing the worker to be directly exposed would be presumably of greatest concern, particularly since the factors of dosage and length of exposure are paramount in human disease factors. Thus, farm workers would appear to have major exposure to animal wastes, hospital staffs and attendants to infective human wastes, sanitation workers handling solid wastes in or outside industry to toxic wastes, and so on. Similarly, family members — especially children — may be intimately exposed to accumulations of solid wastes in and about the home, the risk being proportionate to the hazard in the waste (see sections on safety, human fecal wastes, and animal fecal wastes).

Pathway 5: Water Supply

Solid waste materials which are soluble or suspendible in water may create a potential source of hazard by runoff into surface waters, or by leaching and percolation into ground water or aquifers used for potable water. In addition, the possible change from solid to liquid or suspended, and again to solid state, could presumably cause special concern with evaporation of the solvent and concentration and accumulation of the solid phase (see section on chemical wastes).

Pathway 6: Food Supply

The possibilities for introducing toxic agents or biological pathogens to the human organism through the food–waste cycle are many and complex. Presumably, exposure could result from direct contact, as in the use of containers containing remnants of waste for food transport, from contamination through the agency of biologic vectors such as flies and rodents, and from contamination by waste residues during food processing. Exposure also could come about through contamination of food crops by fertilizers prepared from wastes or prepared foods in markets merchandizing such fertilizers, through contamination of edible fish by discharge of wastes into bodies of water, or by infection of animals used as a source of food.

Pathway 7: Socioeconomic Factors

These will not be analyzed because no definitive studies have been addressed to any aspect of this pathway. Common observation, nevertheless, leads to the conclusion that special interest conflicts, educational and cultural deficits, jurisdictional disputes, and similar factors have caused and will continue to cause delays in correcting deficiencies in solid waste management and, by so doing, create a pathway potentially leading to human disease and degradation of the environment to the lessening of human well-being in general.

C. disease associated with chemical wastes

General

Man uses hundreds of thousands of chemical materials or creates them incidentally in industrial and other activities. Each of these materials, either in its original form or as a breakdown or by-product, will probably appear at some time as a waste. To demonstrate the possible harmful effects on man would require first, that the products be followed, in most cases, through an extremely complex pathway in which numerous changes in chemical nature and concentration may occur; second, that they remain identified in this journey and not confused with materials of the same nature from other sources; third, that they be traced onto or into the human body in a form and concentration that can produce disease; and fourth, that the host be adversely affected thereby.

Barring this, epidemiologic methods are needed to demonstrate convincingly that a disease in a population is of a type to be expected from a specific waste chemical, that this chemical be demonstrated as having access to the population, and that no other source can be similarly implicated.

Attempts at categorization of chemicals for toxicologic purposes have been shown to serve little purpose. Minor changes in structure of a compound can bring about major changes in its biological effects. In addition, the task of itemizing the enormous number of wastes as to their toxicologic potentialities is beyond the scope of this study. Consequently, a postulation of possible pathways for chemical wastes to reach a human body will be described, and attempts will be made to validate parts or all of these routes from the literature. Complete itemization of chemical wastes of toxic or carcinogenic potential will not be attempted. Only those wastes will be discussed that can serve to illustrate points in the postulation, or, if possible, that are known to cause human disease.

The wastes to be considered are those that are normally solids when in a dry state, or liquids of a sufficiently hazardous nature to require special handling.

It is entirely possible that present practices in which toxic chemicals are discharged into water will be proscribed in the future.[267, 272] It will then be necessary to apply special handling procedures to protect the public against new avenues of exposure. In addition, waste chemicals discharged into liquid media may be precipitated by waste treatment processes and become concentrated in the resulting sludge solids, creating other potential risks to public health by pathways to be discussed.

Postulation

Hazardous wastes chemicals can adversely affect the public health via a number of pathways, depending on the method of disposal and the nature of the end product, its concentration, and the effective dose in the population. These pathways are illustrated in Figure 3.

Chemical waste can be disposed of by the following methods: (a) landfill, dumping, surface and subsurface disposition; (b) streams, lakes, etc.; sewage treatment ponds; (c) (as a component of treated waste) fertilizer, soil conditioner; (d) (as a component of treated waste) animal feed; (e) incineration.

Chemical wastes or wastes which contain chemical products may, through handling, produce dust, while their presence in waste undergoing treatment involving aeration may result in their being air-borne through the bubbling action of the treatment process.

As a result of the leaching action of rain, it is possible for chemicals on soil or in dumps, landfills, or wells, to enter ground-water reservoirs and thence human water supplies. When disposed of on land, they can be leached into surface streams or ground water. They can also contaminate water into which they are directly discharged. In this way they can create a risk to humans if the contaminated water supply is used as drinking water, or cause toxic compounds to appear in aquatic life which could then become part of the human food chain.

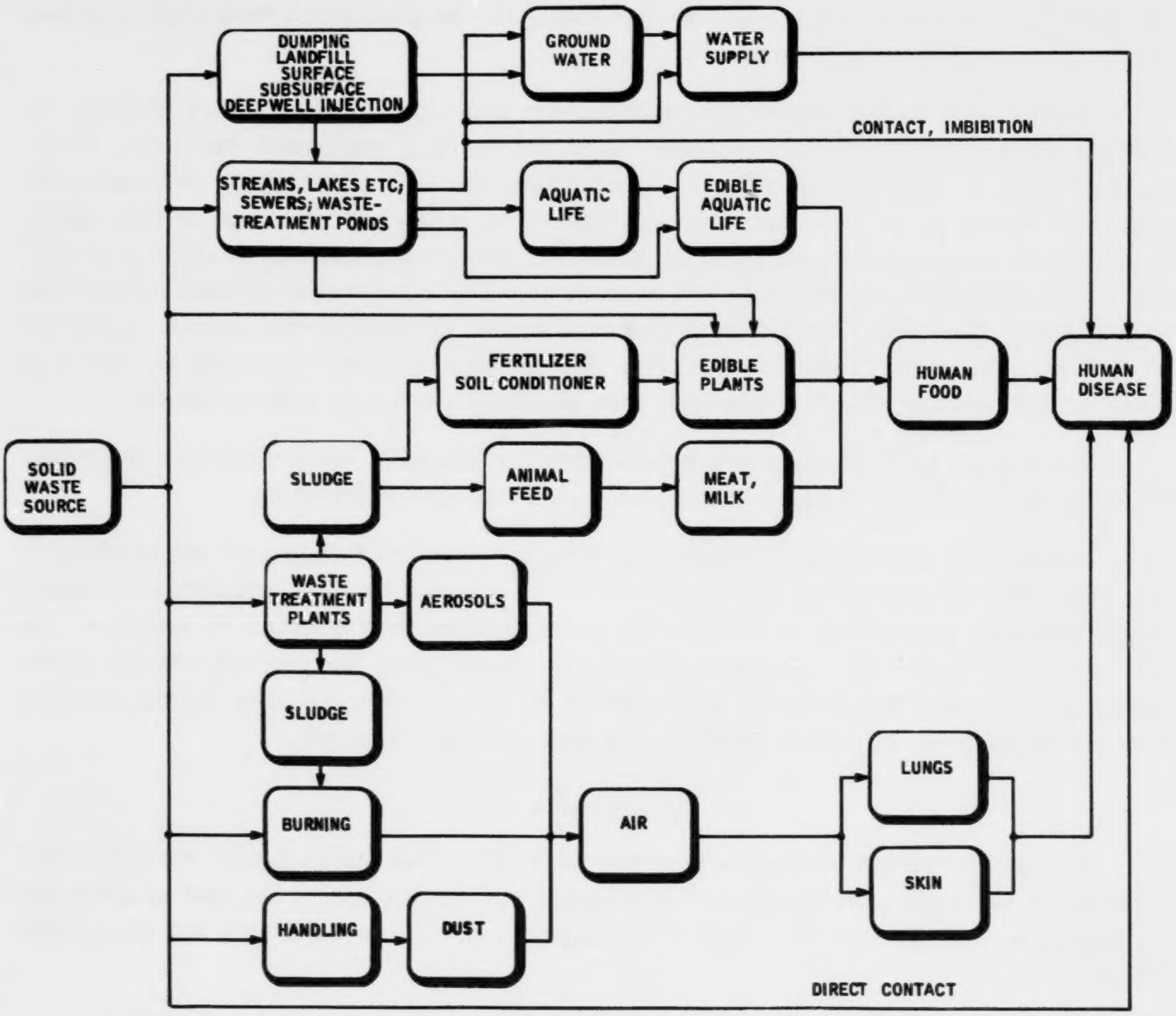
DUMPING
LANDFILL
SURFACE
SUBSURFACE
DEEPWELL INJECTION
GROUND WATER
WATER SUPPLY
CONTACT, IMBIBITION
STREAMS, LAKES ETC; SEWERS; WASTE-TREATMENT PONDS
AQUATIC LIFE
EDIBLE AQUATIC LIFE
FERTILIZER SOIL CONDITIONER
EDIBLE PLANTS
HUMAN FOOD
HUMAN DISEASE
SOLID WASTE SOURCE
SLUDGE
ANIMAL FEED
MEAT, MILK
WASTE TREATMENT PLANTS
AEROSOLS
SLUDGE
LUNGS
BURNING
AIR
SKIN
HANDLING
DUST
DIRECT CONTACT

FIGURE 3. Chemical waste/human disease pathways (postulated).

If chemicals are contained in wastes converted into fertilizers, soil conditioners, or animal feed, they could presumably enter edible plants, meat, or milk and thus constitute a hazard in human food supplies.

Through incineration, they or their by-products can contaminate the skin or the lungs of individuals. In addition, by the handling of wastes, human beings can subject themselves to the toxic action of chemicals through direct contact or inhalation of dust.

Evidence

DISPOSAL METHODS

Land Disposal. The literature deals essentially with five land disposal methods: (a) dumping[2, 70, 136, 214, 216, 217, 231, 240, 243, 290, 291, 636, 640, 714]; (b) landfill[200, 232, 202, 216, 215, 268, 493]; (c) surface deposition[387, 234, 79, 217, 215, 270, 284, 316, 627, 640]; (d) superficial subsurface deposition[200, 70, 202, 255, 270]; and (e) deep well injection.[209, 217, 220, 211, 224, 270, 283, 633, 640]

Water Disposal. The literature is replete with references to discharge of wastes containing chemicals into various bodies of water and flowing streams.[232, 11, 234, 59, 61, 67, 79, 202, 209, 214, 216, 217, 220, 215, 231, 241, 242, 243, 245, 246, 255, 256, 267, 268, 272, 277, 284, 416, 480, 495, 617, 644, 646, 745] The bodies of water include lakes, ponds, oceans, and various lagoons or ponds used for holding or treatment of wastes.

Air Disposal. Wastes are frequently burned.[200, 232, 387, 234, 220, 215, 227, 230, 231, 253, 255, 268, 293, 295, 402, 403, 404, 412, 440, 644] These wastes include all types of municipal wastes, crop residues, and dried sludge from waste treatment processes. Solid wastes also result from burning of primary fuels such as coal, heating oils, gas, and various locomotive fuels such as diesel oils and gasoline.

All of these methods of combustion usually produce particulate matter. The particulates are either discharged into the atmosphere or collected by various means.[23, 34, 240, 293, 294, 296, 298, 297, 299, 411, 426]

PATHWAYS

1) *Soil Contamination.* Some nonbiological wastes are often mixed with other forms of waste for purposes of treatment and become precipitated in the sludge.[63, 136, 138, 202, 207, 209, 215, 224, 227, 236, 241, 243, 244, 245, 254, 268, 284, 402, 495, 710]

One method of disposal of sludge is to use it as a 'fertilizer' or soil conditioner.[202, 214, 215, 216, 254, 268, 280] Thompson *et al.*[268] found 26 metallic components of air-dried sludges in which concentrations ranged from 0.001 to 30 percent.

At this point, the literature becomes highly speculative in regard to disease caused by chemical wastes. Hueper[387] has been perhaps the most outspoken critic of industry in its practices of polluting the soil, water, and air, and has repeatedly warned that these routes of transmission could add to the total carcinogenic load in the population. He states that industrial carcinogenic pollutants may be absorbed by and accumulated "in fruits, vegetables, and food animals using water contaminated with such materials." Rain water could cause soluble chemicals in wastes used as fertilizers or soil conditioners to be made available to plants. Presumably, these could then be taken into the fruits and vegetables or into food animals feeding on forage containing the pollutants.[79] Other evidence is highly circumstantial.[63]

Concern over the possible intoxication of humans by pesticides has led to the finding that some of these agents are accumulated in crops.[646] It is known that pesticide chemicals are absorbed by plant roots and distributed into various tissues of plants.[316] However, similar investigations relating to chemical components of waste *per se* were not discovered. There is evidence, obtained in work on pesticides, that at least some pesticide chemicals are ingested with contaminated food in the human food cycle.[386]

2) *Water Contamination. Surface Waters.* Gurnham[200] states that contamination of streams by toxic wastes creates a hazard to public health: "Injuries to people by such discharges have fortunately been rare, but the killing of farm animals is frequent enough to indicate the danger." This author also states that similar danger exists from food fish which may have absorbed toxic materials from water. However, it has also been stated that there have been no known adverse effects on human health which can be traced to the presence of refractory contaminants in the tap waters of this country.[82]

The literature concerning methemoglobinemia due to nitrates was reviewed to the extent that no case could be traced to pollution due to chemical wastes. However, there is epidemiologic and chemical evidence that this affliction is associated with nitrates and nitrites from water pollution due to biological wastes.[277, 353, 709]

Woodward[14] stated that the presence of goitrogenic materials in some drinking waters "seems beyond question". He speculates that these could be due to the presence of wastes or their degradation products.

There has been some debate on whether certain materials, either by their presence or absence in drinking water, may be associated with certain kinds of cardiovascular disease.[129, 374, 578, 670] However, a panel convened to investigate this possible phenomenon reported that "no causal relationship has been established between the total dissolved inorganic constituents of drinking water and cardiovascular disease."[577]

The possibility that toxic chemicals could enter the human food cycle through their accumulation in aquatic life was investigated. Fish are known to be killed by water pollution, but the mechanisms are poorly understood. In evidence of this is the continued controversy over fish kills allegedly due to pesticides. However, the crucial question for this investigation is whether or not waste toxins enter edible aquatic life through whose consumption humans are affected adversely by these same toxins. There is some indication that oysters can take up carcinogenic agents.[67] Further information concerning the presence or biological amplification of nonradioactive wastes in aquatic life was not discovered.

In the related field of poisoning by pesticides, it was stated[265] that "wide spread use of chlorinated pesticides on the West Coast has not resulted, contrary to often expressed fears, in gross contamination of West Coast fish life . . . ". There was no evidence of biological amplification of these chemicals in this aquatic life.

In spite of the paucity of information concerning this possible pathway, or very probably because of it, concern is expressed: "Surface waters at present serving millions of people are continuously dosed with industrial wastes containing undetermined amounts of unknown chemicals of undetermined toxicity."[267] From their investigations, Borneff and Fischer[46] concluded: "The dosage of cancerogenic substances taken up per person per year is estimated to be of the order of milligrams when surface water is used for human consumption without preliminary carbon filtration."

Hueper and Payne[79] found that extracts of raw and finished water of a river "heavily polluted with industrial chemical pollutants, when subcutaneously injected into mice, elicited the development of . . . sarcomas at the site of injection." These authors also implicate such pollutants as the cause of leukemic reactions and a bladder papilloma in mice.

It is stated that "trace quantities of many chemical, biological, and radiological pollutants are being detected in [the nation's tap] waters in many areas. Although it is not known that their presence is harmful, it is also not known that their presence is safe."[82]

Ground Water. In the proceedings of the 1961 symposium on ground water contamination[745] there is the following comment: " . . . Waste disposal wells and lagoons, leaking chemical storage tanks, and cesspools [were listed] as the most important reported sources of contamination [of ground water]. The most commonly reported inorganic contaminants were salt water, oil-field brines, and sodium chloride, with an occasional report of specific toxic agents such as fluoride, chromium, and nitrate. In most instances, little difficulty was encountered in establishing the particular origin of the more toxic contaminants."[46] The author, W. J. Kaufman, states further: "The inorganic chemical contaminants of ground water differ from organic and biological contaminants in many ways, the most important differences being their indestructibility, the persistence of pollution resulting from their presence, and the great difficulty and cost of their abatement. It is possible to cite numerous instances of small concentrations of toxic inorganics that appeared in ground water and impaired its acceptability for domestic use. It is probable that the major impact of inorganic contamination is not on man's health but rather on his agricultural and industrial enterprises . . . " (p. 43).

Middleton and Walton, writing in this same reference, state: "A wide variety of organic contaminants are reaching ground waters from leaky tanks, lagoons, and septic tanks, or by accidental means. The problem is nationwide, and the reported incidents probably represent a small fraction of actual occurrences. The presence of contaminants has been evidenced by taste and odor, foaming and crop damage. Once the contaminants have entered the ground water, they may travel for long distances and persist for many years." (p. 55).

Also, in these proceedings, Flynn (pp. 71–82) refers to an industrial contamination of domestic water wells by industrial wastes; Deutsch (pp. 98–104) reports on ground water contamination of a domestic well by electroplating wastes, including cyanide, chromium, nickel and copper; Weaver (pp. 104–110) refers to the pollution of ground water by chemicals due to leaching of sanitary landfills. Burttschell *et al.* (pp. 115–117) report ground water pollution by organic industrial wastes; Walter (pp. 121–125) reports on extensive (5 square miles) contamination of ground water by industrial chemicals.

A World Health Organization bulletin [714] states (p. 951) that, in Europe, the chief sources of ground water contamination are domestic sewage and soluble refuse, industrial effluents and soluble wastes, accidentally spilled liquids, saline waters of geographic origin, and sea water. The contamination from domestic or industrial sources is due to pollution of the soil or water courses by wastes without prior adequate treatment. In addition, poor selection of disposal sites has led to pollution of aquifers. Poor construction of wells and poor pumping practices were noted as positive factors in this regard (p. 951). The authors, Buchan and Key, note a number of instances of ground water pollution by industrial wastes.

Scott [11] reported contamination of ground and surface waters by industrial wastes: "Ground water . . . from the test wells . . . was contaminated. . . . Well waters which formerly showed less than 0.1 mg/l iron and manganese now contained 40 to 20 mg/l of these elements. Use of such ground water facilities has necessarily been discontinued. . . ." This author notes the unpredictable nature of flow of the waste from a treatment pond.

Borneff and Knerr [44] reported experiments in which "Particles containing carcinogenic compounds (asphalt, tar, soot, etc.) of the size downward to about 10 millimicrons . . . may reach subsoil water . . . . The behaviour of carcinogenic compounds in soil under the influence of solvents, for example, . . . detergents, cannot be judged definitely until further experiments have been performed." Borneff [45] reported on the development of carcinoma in the mouse only when the carcinogen had been dissolved by surface-acting compounds in the drinking water. However, he states that the solubilization of carcinogenic compounds by detergents contained in waste water, or in surface waters contaminated by waste water, lacked experimental proof. As has already been reported, [257] there is contradictory evidence on the action of detergents in promoting the passage of contaminants through soil.

Potential risk to humans through contamination of underground sources of water supply by cyanides is mentioned. [243] Cyanide wastes are said to have caused fatalities when disposed of in sewer systems. [254]

The potential contamination of ground waters by chlorinated and fluorinated hydrocarbons is discussed. [255] No evidence of actual ingestion of these chemicals by this means was mentioned.

Hettig [260] states that there is potential danger "if subsurface faults allow large amounts of unconverted wastes to contaminate the ground water".

Well water pollution by laundry waste has been reported. [266]

Gross contamination of well waters by oil well wastes and by refinery wastes was reported by Maehler and Greenberg.[270] Morris and Weber[272] also report on ground water contamination by industrial pollutants.

Experimental leaching of ash dumps was carried out by the University of California.[290] This experiment was carried out in an area of very low annual rainfall. A further report[473] states that a sanitary landfill in intermittent or continuous contact with ground water will cause the ground water in the immediate vicinity to become grossly polluted. A report of interest in ground water contamination in relation to refuse is contained in reference 636.

Another study [478] found that, although the practice of dumping acidic wastes has not been permitted in the Baltimore area for 20 or 30 years, ground water in the Patuxent formation in the contaminated area has been rendered practically useless for most purposes. Traces of toxic materials (cadmium and hexavalent chromium) were found.

Miller and others[627] state, "Ponding and ground surface waste disposal continued to be the practices most hazardous to potable ground water supplies and should receive continued scrutiny."

"Wastes too difficult or troublesome for surface disposal are dumped into a deep well where they sometimes pollute useful aquifers pierced by the well." [640]

McKee[644] reports pollution of ground waters by sanitary landfill. Further quotation of similar statements does not appear necessary to establish the fact of ground water contamination by chemical wastes.

3) Air Contamination. Hueper[387] speculates on the contamination of air by industrial wastes. He is concerned particularly with the additive or synergistic actions of such wastes in regard to the total possible carcinogenic load on the population.

One report[231] stated that air pollution through the burning of wastes creates hazards. Of interest to possible pulmonary disease is the statement that, in one study on a large incineration project, 12 percent by weight of the evolved ash consisted of particles measuring less than 5 microns. Lenehan[230] states that a 500 ton per day municipal incinerator will produce 10,000 pounds of fly ash per day unless air pollution control equipment is used. And, while he notes that electrostatic precipitators can remove fine particles with almost 100 percent efficiency, he finds that such equipment is not used because of the cost involved.

Greeley[253] discusses the evolution of gaseous products in incineration of municipal solid wastes. He also notes the potential for air pollution by particulate matter from such sources and states that, insofar as he knew, no electrostatic precipitators had been installed in refuse incinerators in the year reported (1956).

Incineration of waste sludges is noted by Thompson. [268] Although he reports on the finding of some 26 metallic components of these sludges, he gives no data on their presence in airborne combustion residues.

Chambers[293] discusses air pollution by primary solid wastes of industrial source. He lists metallic fumes, and dust containing lead, vanadium, arsenic, beryllium, and nonmetallic elements (fluorine and phosphorus) as being constituents of such wastes. He states that 25 pounds of solid, such as dust, smoke and condensed fumes, result from each ton of waste burned. He also lists the waste solids evolved from combustion of various fuels.

Eliassen[295] states: "The major contributions to air pollution from domestic sources are the products of combustion from space heating and refuse incineration. . . . Refuse disposal is the major source of air pollution from municipal activities. . . . [It] increases with the use of . . . open burning or refuse . . . . " He gives the various amounts of airborne solids produced from different types of waste incineration.

Howe[402] refers to the finding of toxic chemicals in sludge and to incineration of such sludge. No mention is made of any analysis of airborne residues of such incineration.

Rogus[404] states that the burning of refuse accounts for up to 10 percent of total air pollution in urban areas. The contaminants include inorganic gases, organic substances, and particulate matter.

Ashe[408] reports excessive deaths associated with very heavy air pollution among persons recorded as having chronic bronchitis, bronchopneumonia, and other lung or heart disease. The air pollution included "vast quantities of particulate matter and gases of various kinds."

Breslow[409] discusses pulmonary disease, including asthma and emphysema, as possibly associated with air pollution. In regard to emphysema, he states: "We are by no means in a position to draw any sound conclusions about this matter." He also implicates lung cancer as a possible consequence of air pollution.

Nelson[410] discusses "The possibility that the presence of particulates might synergistically potentiate the irritant action of gases on the respiratory system" and states that there appears to be experimental verification of this in animal studies.

Kotin[411] discusses pollution of urban atmosphere by carcinogenic agents. He states: "Carcinogenic materials have been identified in the air in a particulate state that permits their being breathed and deposited on the lining of the lung."

Stenburg[412] states that "excessive discharges of smoke and fly ash to the atmosphere are the recognized by-products of the poor combustion at one time or another identified with practically all types and sizes of [waste] incinerators". He says that customary methods of incineration of wastes remove larger particulates but are not effective in removing particles in the lower micron ranges.

Bush[415] stated: "Particulate material from municipal incinerators amounts to about 4 to 5 lb. per ton of refuse burned. On the basis of particle count, using the electron microscope, this corresponds to 1×10^{15} to 20×10^{15} particles per ton of refuse burned, with more than one half the particles being less than 0.1 micron in size. . . ."

Feldstein[438] states: "The kinds of air polluting products formed during open dump burning, burning of land-clearing debris, burning in single-chamber incinerators and combustion in the automobile engine are quite similar, differing generally in the quantities of each component produced . . . . Greater relative quantities of organic gases, ethylene, particulates, olefins and oxygenates, are produced by poor combustion in single-chamber incinerators than are produced during the operation of gasoline engines." This author also says that "Twenty-four tons of particulate matter are produced during the burning of 2,000 tons of land-clearing debris. Because of the generally poor combustion which occurs in such fires, it is estimated that about 50 percent of the particulate matter is in the size range greater than 50 microns."

Ellsworth and Ballinger[440] state that the burning of automobile bodies has "created such air pollution problems that public opinion has caused the practice to be outlawed in many cities and residential areas."

4) Direct Contact. Material under this heading that is related to sanitation workers will be discussed under *Safety* in a separate section.

The rare reporting of accidental ingestion or inhalation of solid waste materials among the general population does not necessarily mean that occurrences themselves are rare. Any ensuing symptoms could very well be ascribed by the public to other causes. The literature does not provide data on either the frequency or severity of such exposure.

5) Miscellaneous. Although it is known that animal feed is sometimes prepared from waste materials and that the finding of cancer in some animal species has led to the suspicion that such feeds may contain carcinogenic contaminants, there is nothing in the literature relating waste in contaminants to cancer in animals, or to a risk to humans, except in a hypothetical way.

There little in the literature that comments on pesticides and their effects as wastes on man. Such information as exists deals with accidental poisoning of children playing with discarded pesticide containers at home or a rare contamination of food by containers previously used for carrying pesticides.

Discussion

Although there has been considerable speculation in the literature as to potential risk to human health from the various disposal methods used for chemical wastes, the investigator in this study was unable to discover in the literature any reliable evidence supporting a relationship.

There are major barriers to such an attempt. Reference to lack of data is frequent in the literature.

In 1959, Nelson[410] stated: "We have no evidence at this time that systemic diseases from trace quantities of toxins are of significant health concern. It may be that obscure relationships exist of which we know nothing at the present time."

Ettinger[628] says: "There is a vast area of ignorance concerning the toxicology and pharmacology of both synthetic and naturally occurring organic materials, and new materials are being made much faster than the toxicology of known materials is being studied."

Gurnham[200] states that "little is known of the possible cumulative action of metal salts and similar poisons that may lead to general ill health and debility . . . ".

Hueper[387] makes the following statement: "Only very fragmentary information is available as to the potential carcinogenic properties of a considerable number of these agents (carcinogens found in the air, soil, or drinking water)."

Woodward[14] states: "Knowledge of the nature of organic chemical pollutants in water is very sketchy and knowledge of their physiologic significance is even more so." In regard to tap water contaminants, it is said: "Although it is not known that their presence is harmful, it is also not known that their presence is safe."[82] The unknowns of surface water contamination were referred to previously in reference 267.

Thompson and others[268] make reference to the lack of data on the metallic content of sludges.

The need for research on ground water contamination is summarized in reference 745 (pp. 165–215). This need covers many fields, including geology, hydrology, and chemistry, although substantial preliminary studies are available.[745, pp. 7–31; 473, 636, 748] The scope of the ground water research requirement is also evident from a report by Litvinhov[748] on water pollution in eastern European countries (pp. 447–448, 452, 459).

It is stated[714, p. 1000] that "there have been very few attempts to carry out strictly controlled experiments on the purification of polluting matter as it travels through the ground. This is doubtless because of the extreme difficulty — almost impossibility — of accurately simulating natural conditions while retaining the necessary control." Mistakes already made, with resultant long-range pollution of ground water, and their association with ignorance of geologic and hydrologic factors, have been noted in reference 11.

McKee[352] discusses ground water pollution and states that "little quantitative information is available on the actual occurrence and movement of gases through soil and their relation to the quality of water in the upper fringe of the groundwater table."

The uncertainty with regard to drinking water constituents and cardiovascular disease has already been referred to previously.

The following statement appears in reference 640: "Knowledge is scant on many aspects of groundwater contamination."

Weaver[745, p. 109] states: "One cannot help wondering . . . about the unfortunately large number of communities that still resort to uncontrolled dumping. The impact of these practices on ground water and other public health implications involved are cause for concern. We need to know much more about both the geological and climatic characteristics that, along with operational techniques, are so important to short- and long-term effects of degradation and possible leaching of refuse disposed of on land. We also need to have a much clearer picture of conditions as they now eixst in areas where refuse presently is being disposed of by landfilling."

The state of knowledge with regard to air pollution by solid wastes or its incineration is typified by the statement by Dixon[294] to the effect that certain types of air pollutants can cause chronic bronchitis. The exact mechanisms of causation, however, are not clearly understood.

One of the problems in this study was the question of classification of pesticide residues and their relation to crop wastes. A parallel question (that is, when is a waste not a waste) occurs in connection with other wastes which from time to time have been found to have utilitarian value. Another question which is largely unresolved is this: When does the farmer stop being a farmer and become a waste handler? And, in this regard, how does one separate his degree of exposure to pesticides while he is spraying, harvesting, or pruning, from his exposure in the act of hauling away crop waste? This issue is raised, if only to point to the danger of attempts at compartmentalization of environmental health studies.

The quotations given are typical of those relating to the unknowns of waste pathways and quantitative data. However, there exists an equally important hiatus in the knowledge of the disease postulated to be associated with these wastes. One entire segment of medical knowledge that is missing is that involving the effects of small amounts of chemicals of many types ingested or inhaled over a period of many years.

If comprehensive information were available on waste materials and it could be demonstrated that such wastes were entering the population, it would still be extremely difficult to say what relationship they bore to the presence of any disease discovered in this population. Investigators at this time are able only to hint at possible association between contaminants of soil, water, and air, or of materials naturally present in the soil and morbidity.[683, 746, 410, 737, 738, 712] Armstrong[85] summarizes some of the problems to be met in any attempt to trace relationships between soil elements and disease.

Conclusions

The literature makes it clear that there is contamination of soil, water, and air by chemical wastes. Beyond this, there is no factual evidence which permits a conclusion that any human disease is positively related to such wastes. Such information as is contained in the literature on this subject is speculative.

However, certain bits of knowledge have been proffered which compel attention to a possible relationship. For example, the production of large quantities of submicronic particles in the incineration of wastes make it certain that these particles will have direct access to the deep lung structures if they are inhaled. It is known that perhaps 90 percent of inhaled particles of this size are retained in the deep lung tissues. Should such particles contain toxic materials or carcinogenic agents, damage to the lung could occur. In addition, fly ash from incinerated waste contains materials that are known to cause disease in occupational exposures, although there is no evidence that these substances cause pulmonary disease in the general population.

One conclusion reached is that a great deal of contamination is instituted without any knowledge or apparent concern for the possible consequences. This statement applies to all forms of waste disposal.

Recommendations

To establish or negate a relationship between chemical wastes and human disease, an integrated, systematized program of investigation is mandatory — it is mandatory, that is, if a timely result considerate of costs is sought.

This program would be concerned with environments, populations, and their relationships. Under environments it is necessary to determine qualitatively and quantitatively the waste chemical contaminant spectrum. Under populations, it is necessary to define the disease spectrum. (The disease spectrum is not further subclassified at this point because we possess relatively little information on the contribution of environmental contaminants to any disease.) Under interrelationships, it is necessary to relate contaminants and disease in a manner leaving little reasonable doubt as to the relationship. It is convenient to distinguish between studies directed at environments and those at populations. The former are easily separated from medical and toxicologic studies in their execution, if not in other respects.

Since the number of environments is large, preliminary studies are required to determine if there are representative environments permitting a limitation of the total number to be studied, and also to identify those that can serve as controls.

The basic environmental studies finally chosen should develop a useful body of knowledge on the contribution of wastes to environmental contamination.

It should be noted that the 'body of knowledge' thus derived will have little application to the question of disease relationships if there is not parallel study directed at discovery of the effects of waste contaminants on man. Here the field is essentially unexplored. A much greater effort is needed to determine what waste chemicals, in what amounts, over what periods, adversely affect man.

An applicable environmental study — that is, a study applicable to the solution of disease and pollution questions within some reasonable expectant period — must be accompanied by medical, toxicologic, and epidemiologic studies. Expanded animal research is needed in addition to the efforts presently sponsored by the Armed Services, NASA and the AEC, relating to environments of special concern. The pioneering work of Tipton, Schroeder, and others on trace elements and disease requires extension by diagnostic, toxicologic, and epidemiologic investigations of trace elements in selected populations so that some possibility exists for comparing the disease spectrum of these populations with that of waste chemicals in the environment. It remains to be discovered if accelerated exposure and extrapolation techniques can be adapted to estimating the effect of very long exposures to small amounts of contaminants from short-range experiments. Epidemiologic studies of exposed and control populations should be expanded to provide both basic data and the means for estimating the validity of accelerated animal exposure experiments.

Studies conducted piecemeal, or compartmentalized according to some preconceived jurisdictional division, cannot hope to provide the information necessary to timely planning for economic, well-founded pollution control activities. They would also be far more costly and time-consuming in themselves than if an integrated approach were used.

For these reasons, the following program is recommended:

I. Environmental Contamination Studies

Phase 1. Summarization of existing knowledge on waste chemical contamination of the environment and of current and projected research in this field

Phase 2. Requirements analysis
  Types of data
  Types and numbers of environments to be studied
  Personnel
  Methodology and instrumentation
  Jurisdictional responsibility assignments
  Priority assignments

Phase 3. Selection of study plan

Phase 4. Surveys of selected environments

Phase 5. Study analysis and interpretation
  Identification of chemical waste contaminants, sources, and concentrations
  Identification of exposed populations

Phase 6. Report

At any time useful data are obtained, they would be made available to the population study.

II. Studies on the Population

Phase 1. Assembly of existing data on trace elements and disease relationships

Phase 2. Requirements analysis
  Toxicologic data
  Epidemiologic data
  Medical data
  Methodology and instrumentation
  Personnel
  Priority assignments
  Jurisdictional responsibility assignments

Phase 3. Selection of study plan based on existing and postulated environmental contamination data

Phase 4. Conduct of studies

Phase 5. Analysis and interpretation of studies

Phase 6. Recommendations

D. communicable disease

Fly-Borne Disease

GENERAL

THE FOLLOWING LIST of human diseases are said to be transmitted by flies[517, pp. 658-662]: enteric diseases (typhoid, bacillary and amebic dysentery, diarrheas, Asiatic cholera, helminth infections); myiasis, loiasis; onchocerciasis; Ozzard's filariasis; leishmaniasis; African sleeping sickness (trypanosomiasis); yaws; tularemia; bartonellosis; catarrhal conjunctivitis; sandfly fever.

To this may be added as possible fly-borne diseases (see section on *evidence* for references): anthrax; salmonellosis; protozoal infestations; trachoma; poliomyelitis; tuberculosis; hepatitis.

POSTULATION

Flies, according to ecologic factors and species characteristics, are able to carry parasites pathogenic for humans and to transmit them to humans and so cause human infection. Flies are aided or hindered in this by certain characteristics and factors of human origin, among which are socioeconomic and technical levels, cultural mores, and household and personal hygienic practices. When community or personal practices permit accumulations of fly-breeding media, the potential for human infection via fly-borne pathogens is increased. Such media are found in solid wastes of human and animal origin. To support this postulation, it will be necessary first, to associate flies with solid wastes; second, to ascertain that flies are carriers of human pathogens; and third, to demonstrate that flies transmit these pathogens to humans so as to cause infection and disease.

EVIDENCE

Flies breed in large numbers in human and animal excreta [355, 680, 354, 9, 356, 544, 121, 336, 194, 391, 152, 153, 193, 314, 301, 170, 77] as well as in food wastes. [194, 680, 91, 391, 622, 187, 152, 153, 193, 442, 314, 171, 443, 444, 181, 544, 11, 49, 150, 77, 84] They are also found breeding in large numbers in sewage sludge. [688, 544] Species preferences, type of breeding medium, and climatic and micrometeorologic factors have distinct effects on breeding habits, but a number of species are highly adaptable and breed in whatever medium is available. [193, 152, 544]

Nash [680] reported that, as early as 1902, he was certain that "flies bred in deposits of house refuse and manure" and referred to similar conclusions reached by others in 1907 and 1908 (p.142 and p.149). Magy [84] lists the fly breeding media among wastes found in Orange County, California, and 13 species of flies associated with them. The media include crop wastes, livestock wastes, community organic wastes (industrial, food processing, and fertilizer plants), residential refuse, commercial food handling wastes, leaf and litter debris, dead animals, solid wastes (sludge) recovered from sewage treatment plants, domestic animal manures, and miscellaneous wastes (dog and cat droppings, backyard compost piles, spilled garbage, etc.). Hart [475] states that manure management on concentrated livestock farms is a serious problem, principally because of the fly-breeding potential of fresh manure. Taiganides and others [490] state that manure is a health hazard and fly-breeding ground, and must be treated. Garbage discarded by rural inhabitants produced flies (Garrison *et al.*). [150] Dorris [422] found that aquatic fly larvae are produced in large numbers in the sludge of waste stabilization ponds. Wiley [49] stated that a major problem of solid waste storage is fly production.

Hartman [170] referred to the enormous fly production potential of chicken manure: "There was a time when [flies] were so thick that workers . . . had to cover their mouths with rags or handkerchiefs . . . . " In discussing whey solids disposal, Scott [395] said: "Fly breeding nuisances can result from land disposal methods . . . . "

Miles [444] discussed the need to control waste collection and disposal methods to avoid fly breeding; fly production occurs at any point at which flies have access to waste, even in waste incineration plants. An unexpected result of attempts at fly-production control through use of insecticides in privies was discussed by Kilpatrick and Schoof. [301] In untreated privies, few *Musca domestica* were produced; the major species bred was the soldier fly, *Hermetia illucens.* Treated privies showed the reverse: few soldier flies, but large numbers of domestic flies. The former, highly sensitive to the pesticides, were eliminated and the customary consistency of the medium, brought about by their presence, was changed so that the domestic fly, resistant to pesticides, found the medium to its liking.

La Brecque[443] referred to dumps as sources of insects and to half-burned garbage as a source of prolific breeding of flies. Flies will emerge from as much as five feet of uncompacted cover in refuse; their eggs are deposited in the organic matter of the waste prior to collection or at the disposal site if it is not covered rapidly (Black and Barnes).[171] Gotaas[314] said that good fly control is "difficult, if not impossible, when food attractive to flies is composted anaerobically in stacks in warm weather". He stated further that "garbage, faeces, animal manure, abattoir wastes, and tomato and several other food processing wastes, are excellent media for the breeding and development of a large fly population. If adequate control measures are not practiced, particularly when composting manure and faeces, the compost depot will be infested with extremely large numbers of flies and a health hazard almost as serious as that caused by open, uncontrolled garbage dumps will be created."

Black and Barnes[442] stated: "It is generally conceded that one of the most important reasons for burying garbage or mixed refuse is to control flies." However, McGauhey[145] said that burying wastes in the soil may increase crop diseases and insects. Wolf[677] noted the fly-production potential of sludge drying beds and reported the experimental breeding of *Musca domestica* in digested sewage sludge of different ages, pH, total solids, etc.

Webb and Graham[193] referred to enormous numbers of flies, including *Musca domestica,* found in association with a military post garbage dump in Canada. Experimentally they found that "human feces attracted practically all species of flies attracted to the other baits (cooked meat, milk–sugar mixture and chopped fruits) and usually in much larger numbers."

Siverly and Schoof[152] surveyed fly production in Arizona. They recovered *M. domestica* from more than 50 percent of the samples and from 19 of the 21 different classifications of media. "*Muscina spp.,* in common with *M. domestica,* were able to utilize all general categories of production media — excrement, garbage, and miscellaneous types . . . . " The preferences of other species are discussed. In addition, seasonal occurrence as related to species is mentioned as is the year-round adaptability of *M. domestica.* These authors also developed a "production index" by assigning values of 1, 3, and 9 to light, moderate, and heavy infestations of media "whereby the degrees of fly infestation can be quantitatively compared during the four seasons". They stated that " . . . chicken and pig excrement, garbage, melons, and stock feed displayed the highest fly production potential."[153]

Quarterman and others[187] mention dairies, an abattoir, and a city garbage dump as heavy fly-producing sources in a Georgia study. Golueke and Gottas[622] say, "Especially serious are open dumps where flies — flourishing on exposed garbage and breeding countless new generations are paralleled in numbers only by a well-fed rodent population."

Schoof and others[391], reporting on surveys in three U. S. cities and suburbs, make the following statements: "Although numerous media such as fowl excrement, dog stools, sea food wastes, and dead animals yielded a higher percentage of positive samples in relation to the total number of the particular substrate located, the ubiquitous occurrence of garbage in the city overshadowed these as to overall significance in fly production. Dog stools and fowl droppings composed over two-thirds of the total positive excrement samples detected. Because of zoning restrictions, feces of the larger domestic animals were almost totally absent. In the miscellaneous categories, meat, dead animals, and coffee-grounds predominated." "Household garbage (in containers and scattered) was the most frequent producer of positive substrates . . . . " This article is of significant interest in regard to fly production.

McGauhey[91] speaks of the sanitary landfill as "at worst . . . a dismal undertaking conducive to the breeding of rodents, flies, and other vectors of disease . . . . " Coffey and Dunn[465] list food wastes as food for flies. Maier and Baker[178] state that proper disposal of food wastes essentially eliminated all foci of intense fly breeding in a Texas county.

1) Flies and Their Proximity to Humans. If flies are to function as vectors of human disease, it must be demonstrated that they are able to travel to human domiciles from breeding areas and that they have direct contact with man, his food or other articles or substances which can serve as intermediate means of transfer in the passage of disease agents.

As to direct association, certain flies are so well adapted to man and his surroundings that they have become known as 'domestic' species. Others are adapted to man's domestic animals. Other species are occasional visitors of human habitats.

It has been shown that flies travel long distances from breeding areas to human dwellings.[187, 188, 193, 186, 78, 84] Magy[84] noted fly dispersion of up to 20 miles from the source. Under "population pressure" and the odor of attractants, flies migrate readily from breeding site to sources of food and between sources of food.[181, 186, 38, 540, 121, 336, 194, 395, 178, 187, 188, 78] There is also evidence that *M. domestica* is an instinctive wanderer.[188]

Since there is some question that adult flies are contaminated with human pathogens by their breeding medium,[300] contamination by organisms pathogenic to humans must be observed to take place by other means. The wandering characteristic of many flies has been noted above. Specifically, their attraction to both excrement and human foods must have been observed and their contact with other sources of pathogens, and thence with humans, must have been demonstrated if the chain of events from waste to human is to be established.

Some of the earliest observations refer to the visible tracking of lime used to treat human feces in latrines to food served soldiers in mess tents. Other observations attest to this potential or actual mode of transfer of pathogens.[542, 193, 78, 361, 179, 321, 319, 185] Much circumstantial evidence of such transfer is contained in the references given previously.

Direct contact of both bloodsucking and nonbloodsucking flies with humans is common knowledge. Occupational and recreational activities offer many opportunities of contact in areas where flies prevail.[86, 170, 395, 475]

2) Flies as Carriers of Human Pathogens. Recent writers[38, 75] dismiss older studies of the *passage* of organisms through flies as not being sufficiently controlled, but accept older studies attesting to the ability of flies mechanically to carry pathogens. The evidence for both methods of potential transmission is contained in the material on individual disease agents presented in the next section. In summary, however, flies are shown to be capable of both means of carriage, depending on the organism involved. For example, Hawley and others[135] found that bacteria, when fed in sufficient numbers to flies, underwent multiplication and were excreted by them. Knuckles[679] found *Salmonella schottmuelleri* and *S. typhimurium* to multiply indefinitely and throughout the life of the blowfly [*Phormia regina* (Meigen)]. A large literature attests to the external carriage of pathogenic organisms.

3) Flies and the Transmission of Human Pathogens. Although earlier observations of the possible association of flies with disease had been made, the Spanish-American war gave impetus to that impression in this country, and led to great concern among public health officers about sanitary disposal of human excreta. Sternberg in 1899, in reporting "Sanitary Lessons of the [Spanish-American] War," said, "I find that the disease [typhoid fever] was imported by this regiment into its Cuban Camp . . . . ; that it was clearly not due to water-infection, but was transferred from the infected stools of patients to the food by means of flies, the conditions being especially favorable for this manner of dissemination by reason of the close proximity of the picket lines to mess-tents and latrines."[388] In 1901, reporting on the Boer War hospital, Bowlby and others[548] noted that flies seemed to be especially attracted to enteric [typhoid] fever cases. This account reads as follows: "At Bloemfontein the flies were a perfect pest; they were everywhere, and in and on every article of food. It is impossible not to regard them as most important factors in the dissemination of enteric fever. Our opinion is further strengthened by the fact that enteric fever in South Africa practically ceases every year with the cold weather . . . . With the cold nights, the flies disappeared. It seemed to us that the cold weather reduced the number of enteric cases by killing these pests."

Similarly, in 1902, Dunne and Cantab[302] wrote: "The plague of flies which was present during the epidemic of enteric typhoid at Bloemfontein in 1900 left a deep impression on my mind, and, as far as I can ascertain from published reports, on the minds of all who had experience on that occasion. Nothing was more evident than the fall in the admissions from enteric fever coincident with the killing off of the flies on the advent of the cold nights in May and June."

In 1903, Nash[540] wrote: "I feel justified in ascribing the principal role in the causation of epidemic or summer diarrhoea to flies, more particularly the common housefly and the blue-bottle fly. I maintain that flies are the chief agents concerned in carrying faecal pollution to milk and other foods during the summer months." He noted the coincidence of the appearance of flies *(Musca domestica)* and epidemic diarrhea and the death of 13 infants from the latter within three weeks of its onset in the community, and the converse (the rapid abatement of the disease with the disappearance of flies). He was especially concerned with the availability to flies of midden and refuse heaps in the vicinity of cowsheds and dairies, and the contamination of milk which could follow.

Aldridge[355] further indicted flies as transmitters of enteric fever, although he recognized that "we are still without experimental proof." He stated: "It is a well ascertained fact that enteric fever is particularly prevalent where dry methods of removal of excreta, such as pail and earth closets and trench latrines are in use; that is, where flies have ample opportunity of crawling over the excreta, which they do not have in water-closets. There seems to be no doubt that in India, speaking generally, the seasons of greatest prevalence of enteric fever correspond pretty closely with the seasons of greatest prevalence of flies."

Aldridge describes the spread of the disease and the circumstantial evidence matching it with proximity of flies and their access to latrines and messes. He states: "A study of the incidence of enteric fever shows that the stations where there are no filth trenches, or where they are a considerable distance from the barracks, all have an admission-rate below the average, and all but one less than half the average." He also notes the ability of flies to contaminate culture media with bacilli from excreta.

Also, in 1907, Buchanan[360] conducted experiments in which the agents of typhoid fever, swine fever, staphylococcal abscess, pulmonary tuberculosis, and anthrax were successfully transmitted to culture media by flies, and noted the death of a guinea pig inoculated with the culture of the tubercle bacilli thus obtained. He made the following summation: "The experiments conclusively show that flies alighting on any substance containing pathogenic organisms are capable of carrying away these organisms in large numbers on their feet and of depositing them in gradually diminishing numbers on surface after surface with which they come in contact. They further serve to demonstrate the necessity for the exercise of stringent measures to prevent the access of flies to all sources of infection and to protect food of all kinds against flies alighting on it."

In 1908, Klein[303] reported that, in addition to a great number of *B. coli communis*, "limited numbers [of] a species of microbe which is not distinguishable from *B. typhosus*" were found in cultures grown from minced flies. The flies had been obtained by Dr. Heywood Wilshaw from a locality in which enteric (typhoid) fever had occurred.

Nash[680] in 1909 referred to Nuttall's 1899 summary of the literature to that date dealing with flies in relation to disease and covering 350 references dating to the 18th century (p. 160). Flies were incriminated as carriers or transmitters of anthrax, plague ("carrier state conclusively proved"), typhoid, tuberculosis, leprosy, cholera, and diarrhea (pp. 160–161). Nash blames the fly and calls attention to the rapid decrease in incidence of disease when frost kills off the flies so that "no 'carriers' are available" (p. 162). He points to the danger to infants from fly-borne disease (p. 164).

Major Faichnie, writing in 1909 in the *Journal of the Royal Army Medical Corps*, reported this incriminating data on flies: "Caton Jones pointed out that since a raid on flies had been commenced in Nasiribad, in 1904, the enteric fever rate there had very much diminished, and that the results obtained were partly due to a better system of trenching the night soil, by which the breeding of flies was prevented." He wrote that "In Mhow, there was also a sudden diminution in enteric fever in 1907, which has been maintained ever since. This diminution coincides with the inspection of the station by Surgeon-General Trevor, who found the trenching grounds swarming with flies. Since then . . . for eighteen months scarcely a fly has been bred there." He felt that typhoid inoculations, which did not include all personnel, could not account for all of the reduction in cases and noted that the water supply was pure. Further, "One station may swarm with flies, bred only from the excreta of cows and horses, and yet have not enteric; while another place, where there are very few flies, but where these are bred from human excreta may have an epidemic. . . .It is the breeding ground that constitutes the danger . . . . "[354]

Ainsworth, [199] in the same journal in the same year, had this to say: "I readily admit that the operations recorded and the arguments advanced in this paper are open to the objection that they afforded by scanty data upon which to base so important a conclusion that the house-fly is frequently the intermediary and probably by far the most common intermediary, in the propagation of . . . enteric fever. Nevertheless, . . . there is an isochronism . . . between the advent of the house-fly in Poona and the seasonal prevalence of enteric fever, which is highly significant and at least suggests that a prima facie case has been established for further investigation." He demonstrated that seasonal heat and moisture "combined with suitable breeding media, will of a certainty produce flies." He concluded that his charts on fly/enteric coincidence are sufficient proof of the fly's role as intermediary and the acceptance of the disease as "probably" fly-borne.

Dr. James Niven, [9] in 1910, noted that the "social condition" related to the health of infants had much to do with their death from summer diarrhea. He made the following observation: "The summer wave is not due to dust . . . There is nothing to support the view that the infective organisms are of animal origin, and the connexion between privy-middens and diarrhoea goes far to prove the contrary. The disease becomes more fatal only after house flies have become more prevalent for some time, and its fatality rises as their numbers increase and falls as they fall. The correspondence of diarrhoeal fatality is closer with the number of flies in circulation than with any other fact." He stated also that " . . . no other explanation even approximately fits the case." In association with fly prevalence and numbers during summer diarrhea epidemics and the ability of flies to carry bacteria, Dr. Niven felt that it was "unnecessary . . . to multiply references".

In this country, Ridlon[356] in 1911 reported on typhoid fever in Charlestown, West Virginia, and felt that "the most probable source in 5 cases was from flies. These cases were located within 200 feet or less of other cases where the disinfection of stools was inefficient, where there were no screens, and where the abundant flies had free access to both dejecta of patients and the food." He notes that fly prevalence can be "greatly limited by proper care of their breeding places, including stable manure, household refuse and garbage." He did note the possibility of numerous other sources of infection, including public water supply (highly contaminated), food, ice, and personal contact, but discounts water supply in the cases at hand. He ascribed ten cases to personal contact.

In 1912, Cox and others[678] reviewed the literature dealing with bacteria carried by the housefly and reported their own investigations on this subject. They found that flies coming from "insanitary or congested areas of the city" (Liverpool) carried from 800,000 to 5,000,000 "aerobic bacteria" per fly, while flies from the "more sanitary, less congested or suburban areas" carried 21,000 to 100,000 per fly. Similarly, the number of "intestinal bacteria" carried were greater in the former (from 10,000 to 330,000,000) as compared to 100 to 10,000 in the latter.

It was found[678] that "Pathogenic bacteria and those allied to the food poisoning group were only obtained from the congested and moderately congested areas and never from the suburban areas." The authors identified 126 strains of bacteria, among which were streptococci, staphylococci, sarcinae, *B. pyocyaneus*, several of the colon groups, including *B. coli communis*, salmonellae, "Morgan's infantile diarrhoea group", and miscellaneous groups.

Flies were soon incriminated in other diseases. Le Boeuf[358] in 1912 found flies *(Musca domestica)* heavily contaminated with Hansen's bacillus after their feeding on leprous ulcers. He thought that this fly was possibly an important factor in the spread of leprosy through deposition of bacilli in wounds of healthy individuals.

In 1912, Rosenau[369, p. 1593] announced the "apparent" experimental transmission of poliomyelitis from sick to well monkeys by the bite of the common biting fly, *Stomoxys calcitrans.*

Also in 1912, Anderson and Frost[368, pp. 1733-5] stated that their experiments showed conclusively that "in confirmation of (results) announced by Dr. Rosenau," poliomyelitis was transmitted to monkeys through the agency of the stable fly *(Stomoxys calcitrans).*

Brues and Sheppard in the same year felt that this fly *(S. calcitrans)* was implicated epidemiologically in the transmission of poliomyelitis (pp. 305–324).[370]

In a recognized classic, *Flies in Relation to Disease: Non-Blood Sucking Flies*, Graham-Smith summarized the evidence at that time (1913) in the following way (p. viii): "Far reaching conclusions founded on insufficient data at present available can fulfill no useful purpose . . . . It may be claimed, however, that a very strong case has been made out for the thorough investigation of the relation of non-biting flies to disease."[121] He stated also, "In order to determine with any degree of certainty the part really played by flies, we need more particularly a large amount of epidemic evidence such as would be afforded by changes in disease incidence following the control of the fly nuisance. At present there is very little such evidence and until recently there was none. Vague surmises have been plentiful, but trustworthy observations few" (p. ix). He then proceeds to document existing information, pointing out the difference between fly contamination with laboratory cultures of organisms and natural contamination, criticizing earlier bacteriologic methods in the identification of bacterial species, and, especially, epidemiological methods (p. 125). However, on page 126, he states that " . . . in a few instances the evidence appears to be conclusive." He notes the problem of identification of the agent of typhoid fever (p. 129). He states, nevertheless (p. 146), that "The reports relating to military camps in war time show very conclusively that flies are under those conditions the principal agents in spreading the disease [typhoid fever]." [121]

Graham-Smith is of the opinion that, as to dysentery, paratyphoid, and food poisoning " . . . no instances of infection by flies have yet been recorded" (p. 148). Of summer diarrhea, he says (p. 172) that "The epidemic and bacteriological evidence is so suggestive . . . that an attempt to definitely settle the connection between flies and summer diarrhea by preventive measures against flies in a selected area seems now justifiable." He feels further evidence for the fly–disease association is needed for cholera (p. 175), that the relation to tuberculosis is uncertain (p. 179) as it is in regard to anthrax (p. 186). He apparently accepts the evidence (Howe, 1888; Nuttall and Jepson, 1909) for the spread of polio, but says, " . . . up to the present, we have little knowledge of what part [flies] play in the dissemination of disease" (p. 190), and extends this conclusion to include smallpox, tropical sore, trypanosomiasis, and yaws (pp. 190-194). [121]

In 1913, Brues repeated his claim that epidemiologic and experimental evidence strongly implicated flies in polio transmission. [189, pp. 101-110] Terry, in 1913, in a study of typhoid fever in Jacksonville, Florida, stated: "I am fully aware that the facts above stated do not furnish all the requirements of strictly scientific proof that our typhoid fever was for the most part fly-borne, but it would appear that this was the case, as the only measures we have made use of to reduce our typhoid rate have been directed against this insect and the only change in sanitary conditions throughout the city has been the fly-proofing of the privies. I feel that we are justified in attributing the major portion of our cases, prior to the enforcement of privy law, to the house fly." [336]

In 1914, Mitzmain [359, pp. 75-77] reported the successful transmission of anthrax from animals just dead from the disease to guinea pigs through the bites of stable flies *(Stomoxys calcitrans)* and horseflies *(Tabanus striatus)*. The organisms were recovered from the vectors.

In another classic, *The Housefly*, C. Gordon Hewitt [194] discusses the habits of flies in relation to their possible association with human disease (pp. 89-96), the housefly in regard to breeding media, and other fly species in relation to human domiciles (p. 201). He assembles evidence on flies as carriers of pathogens (swine fever, p. 204; intestinal infection, p. 200; eye diseases, p. 213; anthrax, p. 201; and fly contamination by microorganisms (pp. 218–219).

In 1921, Root [304] reviewed experiments dating back to 1913 on the carriage by houseflies of intestinal protozoa. In his own experiments, he found that cysts of Giardia remained viable in the fly for 16 hours, that cysts of amoebae remained alive for 48 to 50 hours in the fly and that cysts of Chilomastix were viable up to 80 hours. He further commented that "If a fly containing a cyst is drowned in water, milk, soup, or other liquid food, the cyst will live still longer, about a week, and there seems to be a possibility that human beings might be infected by swallowing such drowned flies (pp. 131-132, 139, 150–153).[304]

Hall, in 1929, implicated flies and other arthropoda in the transmission of helminths (pp. 1, 2, 64, 69-72).[305]

Gill and Lal established in 1931 that cholera vibrios were capable of surviving in the fly for a period of at least five days. Furthermore, the fly was shown to be capable of infecting food by its feces. They felt that, in hot countries, insect transmission was of predominant importance and that "One of the most important, if not the most important, method of controlling cholera is the provision of an efficient system of sanitary control, more especially in the collection, removal, and disposal of night soil and refuse."[361]

In 1935, Craig[318] by various epidemiologic arguments supported the transmission of amebiasis by flies. He discussed this in connection with the use of night soil on gardens (pp. 46-47), and discussed further an epidemic of the disease observed and believed by him to be vectored by flies (pp. 56-58).

In 1938, Pokrovsky and Zima[179] stated that "there is not the least doubt that flies are carriers of helminth eggs, chiefly on the feet." Their experiments showed that flies could carry the eggs of *Enterobius vermicularis*, of *Diphyllobothrium*, of *Ascaris*, of *Hymenolepis* and cysts of protozoa. They found 47 percent of flies captured in food shops to be infected by such eggs. They emphasized the need for health education to correct this situation.

In 1939, Manson-Bahr stated that "The evidence against the house fly is fairly complete" in association with transmission of dysentery bacilli. He noted the coincidence of epidemics of house flies with epidemics of bacillary dysentery and the decline of the disease incidence during periods when flies were rare. His conclusion from the works of others was that, to a limited extent, amebiasis could also be transmitted by flies. He also noted the possibility that human feces could serve to spread the disease through contamination of vegetables and fruit in those areas where night soil was used for agricultural purposes. He felt, however, that cholera was mainly a water-borne disease.[321]

Claphan, in 1939, showed that flies could serve as intermediate hosts of the nematode *Syngamus trachea*, a cause of "gapes" in chickens.[192] While this nematode is apparently not a human pathogen, the work implicates flies as carriers of disease agents in general.

In 1940, Craig and Faust[319] stated that "the contamination of food and drink by droppings of flies that have fed on material containing the cysts of *Entamoeba histolytica* is an important method of transmission where flies are numerous and foodstuffs are unprotected from these insects. This method is most important in military camps, work, or recreation camps, and wherever large numbers of individuals are gathered together and flies are prevalent." In regard to leishmaniasis, they stated "Today the only insects considered to be transmitting agents are flies of the genus *Phlebotomus*." However, they noted the small number of successful attempts in the experimental transmission of the disease by this route. They commented that all attempts to that date to produce infection in man by bites of the sand fly, *Phlebotomus papatasii*, have been negative, although it was felt that oriental sore, a form of leishmaniasis, was believed to be most commonly transmitted in nature through the bites of this fly. The fly was proven experimentally to be contaminated by the organism responsible for this disease. It had been found possible to produce the sores in man by inoculating material containing these agents which had developed within the fly, although "the crucial experiment of transmitting infection by the bite of the fly has not been successful." Craig and Faust also noted the transmission of *Trypanosoma gambiense* by the bites of flies of the genus *Glossina* (tsetse flies). However, flies were not implicated in the transmission of *Trypanosoma cruzi*. The authors did not implicate flies in the transmission of *Balantidium coli* or *Ascaris lumbricoides*. Similarly, flies were not indicated as carriers of the eggs of trematodes pathogenic for man.

Craig and Faust discussed the flies which are considered capable of transmitting pathogens to man and implicated the common housefly, *Musca domestica*, in the enteric infections — typhoid fever, salmonellosis, bacillary dysentery, cholera and amebic dysentery. They stated that the control of houseflies was responsible for the decline in typhoid epidemics. They equated the domestic flies with the spread of the pathogens of tuberculosis, plague, tularemia, anthrax, brucellosis, typanosomiasis, leishmaniasis, acute catarrhal conjuntivitis (pinkeye), yaws, and trachoma.[319]

Sabin and Ward[366] demonstrated in 1942 the presence of poliomyelitis virus in batches of flies taken during epidemics of this disease in two major cities in the United States. They concluded that there was no doubt that flies were carriers of the agent, but, in these experiments, were unable to determine where the flies obtained the virus. They felt that the chief reservoir of infection lay in human excreta and that spread of the disease by insects was possible.

Ostrolenk and Welch[357] demonstrated in 1942 that a potent strain of *Salmonella enteritidis* could survive the duration of the life of experimental flies. They stated: "In our preliminary studies of this problem we were able to demonstrate that flies are an extremely potent source of pollution organisms, particularly in those food plants where little attention is paid to sanitation and where the food is prepared for the consumer without a final treatment to destroy these organisms." They found that the organism was transmitted easily and rapidly through several populations of flies and that these flies contaminated all surfaces with which they came in contact. They found that the fly carried the organism both externally and internally. They were able to demonstrate transmission of food poisoning bacteria from flies to mice, and a retransfer from infected mice to flies.

In 1943, Bang and Glaser[183] demonstrated the recovery of mouse-adapted human poliomyelitis virus from adult biting flies infected by feeding on mice.

In 1943, Trask and others[159] recovered poliomyelitis virus from samples of flies collected within areas of epidemic poliomyelitis. The method of demonstration was the infection of Java monkeys by the intranasal and intra-abdominal inoculation of material obtained from the flies.

In 1947, Roberts[185] concluded that while flies were proven carriers of the cysts of *Entamoeba histolytica,* flies in the urban United States were of little significance in the transmission amebic dysentery. They stated, however, that "in rural areas . . . particularly in the tropics, flies may prove of greater importance, for, under such conditions, not only do they tend to occur in greater numbers, but, owing to lack of sanitation, they have readier access to infected excreta and to human-food supplies."

In 1947, Melnick and others[176] carried out a fly-control program in association with investigations on fly-borne poliomyelitis. They stated: "A temporary reduction in flies was achieved . . . Under the circumstances, which were not ideal, there was no effect on the poliomyelitis control." They pointed out why the results could not be considered conclusive. The experiment, however, was important in that it represented one of the first efforts which used modern insect control methods in the study of the epidemiology of suspected fly-borne disease.

In 1947, James reported on the fly species that cause myiasis in man. He concluded that such species were "almost exclusively nonbloodsuckers in the adult stages . . . ".[8]

In 1948, Philip[378] was able to recover the agent of Q fever from houseflies *(Musca domestica)* caught at large in an animal room in which Q-fever-infected animals were kept. He was unable to transfer the infection between infected and noninfected guinea pigs to which houseflies had free access. However, the test organism was recovered from houseflies allowed to feed on infected material and shown to cause Q fever in injected test animals.

In 1948, a second highly important experiment was conducted by Watt and Lindsay,[38] using modern insecticides as agents of fly control. These authors were critical of past observations on the fly as a vector of disease and stated: "Common assent has for years ascribed to the fly a major role in the spread of enteric infections. Evidence for this belief was incomplete and did not permit an evaluation of these insects as disseminators of disease." They used treated and untreated towns, and studied the dissemination of *Shigella* and *Salmonella* as dependent variables. They then reversed the treatment and control role of the towns. They concluded with the following observation: "In the area of high morbidity studied, a significant reduction in the amount of infection, disease, and death resulted from the degree of control established. The effect on *Shigella* infections was greater than on infections with the *Salmonella* group of organisms."

In 1949, Pipkin[27] stated: "Although the experiments of earlier workers are not to be discounted, it is thought that modern cultivation and techniques offer more critical and exact methods of evaluating viability [of cysts of *Endamoeba histolytica*]." His own experiments led him to conclude: "The passage of viable cysts of *E. histolytica* in the fly vomitus 64 minutes after initial ingestion and in feces four hours and 20 minutes after ingestion offers a potential natural method of transmission under conditions commonly occurring in backward and rural sections which might possibly explain community amebic infections."

In 1949, Melnick[365] was successful in recovering poliomyelitis virus from flies collected during an urban epidemic and transmitting the disease to monkeys inoculated with the virus from these flies.

In 1951, Hawley and others[135] conducted experiments which consisted of feeding *Musca domestica*, the common housefly, with known numbers of *Escherichia coli, Salmonella schottmuelleri*, and *Shigella dysenteriae.* They found that flies that were fed fewer than 1,000 of the various species of organisms in a single feeding apparently did not pass the bacteria in their stools, but with increasing numbers of the bacteria fed, definite multiplication did take place within the flies as indicated by their recovery in the stools in greater numbers than were fed. These investigators found that the excretion of the organisms remained at a high level for at least six days.

In 1951, West in his book *The Housefly*[90] stated (page 266): "There is at the present time acceptable laboratory proof of the transmission of approximately 30 diseases (or parasitic organisms) by *M. domestica* and related forms." The author implied that typhoid fever, paratyphoid fever, cholera, bacillary dysentery, infantile diarrhea, anthrax, conjunctivitis, tuberculosis, and leprosy are transmitted to human beings by flies, although no factual data of an epidemiologic nature are presented. Similarly, he implicated viral disease, trachoma, polio, spirachetal disease, yaws, protozoan disease, amebic dysentery, and giardiasis. However, he stated: "Flies are rarely the sole transmitting agency . . . and in most cases, their role, however, important, must logically be considered subordinate" (p.272). Most of his discussion is with regard to the ability of flies to transmit disease organisms, and there is essentially no epidemiologic evidence presented or referred to.

Gerberich[184] reported in 1952, that chickens fed on infected flies were experimentally infected by *Salmonella pullorum.* He concluded that his data established the housefly as a vector of *S. pullorum,* and thus increased the potential area of bacterial dissemination to that of the range of the flight of the housefly.

In 1953, Melnick and Dow[363] reported further on flies and poliomyelitis, and on Coxsackie virus and flies. They found that poliomyelitis virus was present in flies in the absence of clinical cases in the town investigated, and concluded that poliomyelitis infections were common in spite of the absence of paralyzed cases. They state: "Intimate contact between persons is of unquestionable importance in the transmission of poliomyelitis. A problem which still remains unsettled is to what extent this added dissemination of virus by flies contributes to the marked seasonal pattern of the usual poliomyelitis outbreak." They remarked that there was no apparent true host–parasite relationship between polio and Coxsackie viruses and flies. They found that there was an apparent correlation between poliomyelitis virus recovered from flies and in privy specimens collected in the same area. Furthermore, they were able to transmit poliomyelitis from virus found in flies to monkeys. They concluded that other studies are needed to evaluate the importance of flies in the transmission of poliomyelitis during epidemics.

In 1953, Francis and others,[364] reporting on the same 1948 epidemic of poliomyelitis referred to in the previous reference, stated that there was a marked correlation between the presence or absence of poliomyelitis virus isolations from privy specimens and from the flies tested as reported by Melnick (above).

In 1953, Morellini and Sacca[329] refuted a number of previous claims or observations associating flies with the dissemination of tuberculosis. Also in 1953, Corbo[92] reported a seven-year study on the trend of infant mortality due to gastrointestinal diseases in an Italian province. He observed that the mortality curve dropped considerably when houseflies were eliminated by the use of insecticides. He found also that where resistance of the insects to insecticides was present, infantile mortality due to these diseases was not reduced.

In 1953, Lindsay and others[76] concluded from a study that "During effective fly control in an area of moderate diarrheal disease morbidity, the prevalence rate of *Shigella* infections and the morbidity rate from diarrheal disease were significantly lowered."

In 1954, in a continuing study of pathogenic human viruses and flies, Melnick and others reported on a seasonal variation in which Coxsackie viruses appeared in some specimens obtained in the summer and fall, but disappeared in the winter and spring. Melnick found that Coxsackie viruses were recovered more regularly from sewage than from flies. The virus was obtained from flies in residential areas not otherwise revealing any source of contamination.

In some instances, the virus was present in residential areas but not at the sewage disposal plant, the virus apparently having failed to survive the period of transportation in the sewerage.

In 1956, Lindsay and Scudder[75] stated: "The nonbiting flies are probably more responsible for the transmission of the causative agents of enteric bacterial infections of all types than are biting flies, including mosquitoes." These authors also said: ". . . In many tropical and subtropical areas devoid of modern sanitation, the principal factor influencing the spread of disease agents by flies and other means is the degree to which the etiologic agents themselves are present. This is also true in many marginal areas with inadequate sanitation and may likewise occur in areas of normal high standards during temporary disruptions of sanitation facilities following disaster." Because of the poorly developed status of bacteriological techniques and taxonomy in the years before World War II during which most of the studies on passage of organisms through flies was performed, Lindsay and Scudder felt that controls on those experiments were inadequate and that the organisms involved are now impossible to identify in modern terms. They state, however, "By way of comparison, the earliest work on the ability of flies to serve as mechanical vectors is probably as significant today as when it was done." These authors refer to important studies on shigellosis carried out by the United States Navy in Cairo, Egypt. In these studies, infant mortality declined and recurred impressively in connection with a decrease and increase in fly population associated with insecticide control efforts. They also refer to an unpublished demonstration by Dow of the feasibility of transmission of the organism responsible for pinkeye by the gnat Hippelates, and to a relationship between school absenteeism, "sore eyes", and numbers of this gnat. Murihead-Thomson was quoted as reporting in 1954 on an association between a fly species and epidemic conjunctivitis in India. Also cited were studies by Satchell and Harrison which "pointed most convincingly to the transmission of (yaws) in part by wound-feeding flies." Lindsay and Scudder state further that "The causative agents of some 30 diseases in all have been associated with, or demonstrated by, laboratory techniques to be capable of transmission by nonbiting flies. Without indicating the degree of importance, it may be said that flies constitute one mode of transmission for the agents of such diseases as cholera, and various protozoan and helminth infections." They said also that "The diverse findings shown indicate the need for systematic studies of the conditions that promote or inhibit the survival, multiplication, and passage of various microorganisms."[75] Smith,[332] in 1957 stated that while claims (Rosenau, 1912; Anderson and Frost, 1912) had been made that biting flies could transmit poliomyelitis virus, others (McFarlan *et al.*, in 1946) in fairly recent years were unable to confirm this either experimentally or on epidemiologic grounds. The possibility of a mechanical transmission to man by infection of flies feeding on feces had attracted more interest. However Smith said: "There is no evidence that it is of any epidemiological importance."

In the book *Bacterial and Mycotic Infections of Man,*[682] published in 1958, flies are not mentioned in relation to the epidemiology of anthrax (pp. 339–340), but are implicated as possible transmitters of salmonellosis and typhoid (p. 387) and bacillary dysentery (p. 397) through food contamination. In the same text, deer flies are reported as transmitters of tularemia (p. 432). No mention is made, however, of any association with domestic fly species. No association of flies is made with swine erysipelas and erysipeloid in man (p. 461) in this book. Spread of cholera is attributed to flies (p. 468).[682]

In 1958, Schliessmann and others[545] reported on a three-year study, completed in 1957, on the relation of environmental factors to the occurrence of enteric diseases in mining camps in Kentucky. These authors found in their investigation that housefly abundance was not significantly correlated with morbidity or Shigella prevalence. They stated: "Lowest rates of reported diarrheal disease, Shigella-positive cultures and Ascaris-positive stools were recorded among study families served by complete community sanitary facilities . . . The highest levels of the three indexes were reported from populations living where community sanitation facilities were entirely lacking. Individuals living in homes provided with inside piped water and privy excreta disposal reported approximately twice the diarrhea and twice the Shigella prevalence and over three times the Ascaris infection rate experienced by individuals using piped water and flush toilets."

Of interest was a further factor — namely, that the population groups using privies showed lower infection rates of Ascaris and Shigella when water sources were inside the house than did those groups whose water source was outside. Lower rates also seemed to accompany the availability of installed bathing fixtures. Schliessmann and others felt that their results strongly supported "the premise that incidence of acute infectious diarrheal disease may be reduced significantly through selective modification of specific environmental factors within communities without regard to etiologic or sociologic differences." It is concluded that "specific environmental improvements, based on a knowledge of local deficiencies, will invariably effect significant reduction in enteric disease."

In a World Health Organization report in 1958 [331] it is stated that the "inadequate and insanitary disposal of infected human faeces leads to the contamination of the ground and sources of water supplies. It often affords an opportunity for certain species of flies to lay their eggs, to breed, to feed on the exposed material, and to carry infection." The report also states: "In different parts of the world, different modes of transmission may assume various degrees of importance: In some areas, water, food, and milk may be most important; in others, flies and other insects; and, in still others, direct contact may assume a major role. What is most probable is a combination of all . . . The technical objective of sanitary excreta disposal is therefore to isolate faeces so that the infectious agents in them cannot possibly get to a new host." The report states also that "In temperate climates, excreta-borne diseases are usually more prevalent during warmer months when flies are more numerous and most active". [331]

In 1959, Greenberg, [182] presented arguments as to why early investigations dealing with the transmission by flies of pathogenic organisms were unreliable. He reports on the variable success in the recovery of different species of Salmonella and of *Shigella flexneri* from fly larvae, pupae, and adult houseflies. When organisms were recovered, he noticed a decrease between those found in maggots and those in the pupae stage. He found that a small number of the pupae were sterile or retained only a few bacteria. In a second paper, Greenberg noted [300] that both the transmission of *Salmonella typhi* or *Shigella flexneri* from the larva to the adult housefly is consistently unsuccessful if the pathogen is introduced into a contaminated larval medium. Under the same conditions, limited transmissions occur with an organism such as *Salmonella enteritidis.* He experimented with laboratory Chemical Specialties Manufacturers Association (CSMA) broth, human feces, garbage, and horse manure as fly-breeding media. Greenberg stated: "Some flies retained only a single species of bacteria whereas the majority of flies harbored an assortment of species. These species are widely distributed saprophytes present in the feed, and feces of horses and other animals. . . . The inability or limited ability of the pathogens to propagate under competitie conditions in the various larval media likewise prevents or limits their retention by the host. The author states "It appears that enteric pathogens are destroyed in the very environment required by maggots for their development." In considering the infectivity of the newly emerged adult fly, he continues as follows: "Typical associates of newly-emerged houseflies are Proteus, Pseudomonas, and various Coliforms. Justification for categorizing them as potential human pathogens lies in their increasing implication in diarrheal diseases, especially of infants. Certain of the true pathogens, for example, *S. enteritidis* and *S. paratyphi B.*, may also be present in the adult fly, but their numbers would probably be quite low considering the relatively low counts of most newly-emerged flies." He stated also: "On the basis of dosage delivery, the risk of human infection from such a fly is probably slight." He further states that "It should be emphasized that the successful transmission of pathogens is more often the exception than the rule when normal contamination of the larval medium is permitted. This has been shown for polio and other viruses; for *Salmonella pullorum;* for *S. typhi;* and *Shigella flexneri;* for several species of Leptospira; and for Toxoplasma." For this last quotation, he refers to the works of other authors.

Medical Entomology[195] contains very little information concerning the fly–human disease relationship not already referred to above. The author does comment on the nuisance value of flies as pests which interfere with man's activities.

Chandler and Read in 1961 reported a case of myiasis due to the fly *Psychoda.*[689, p.786] This fly has been found breeding in sewage sludge[688, pp. 544–555.] The authors associate the sandfly (*Phlebotomus*) with sandfly fever and leishmaniasis (pp. 672-673), midges (Heleidae) with filarial worms of man (p. 678), and hornflies (*Siphona irritans*) with serious losses in domestic animals (p. 693). They associate stable flies (*Stomoxys*) with similar effects on man's meat and dairy animals, as well as with trypanosomiasis, anthrax, conjunctivitis in children, and the transmission of domestic animal parasites (pp. 694-695). They ascribe to the housefly the ability to transmit "filth germs, especially those affecting the eyes and the alimentary canal," gonorrheal ophthalmitis, bacillary dysentery, and other enteritides (p. 696). These authors also relate the eye flies (Chloropidae) to human eye infections and yaws (pp. 708-709). Some flies which breed in human and animal wastes are said to be the cause of some forms of myiasis (pp. 767-794). Other flies, while related to disease, cannot be related to waste because their breeding or feeding habits do not require waste.[688]

In 1962, Scott and Littig[395] discussed flies as nuisances. They stated: "Domestic flies can be a serious threat to individual efficiency. In a fly-infested office the senior author has observed employees spending over fifty percent of their time swatting and driving away flies. Biting flies disrupt picnics and other recreational activities as well as the pioneering efforts of mankind." The authors state that deer flies, horseflies, sand flies, punkies, and other biting flies attack man and cause him great discomfort. They state also that the stable fly can bite severely and the black fly can attack in such numbers that they kill the victim (whether the victims are small or large numbers is not stated). The invasion of tissue fly larvae (myiasis) is noted. These authors also state that domestic flies can mechanically transmit typhoid, paratyphoid, cholera, bacillary dysentery, infantile diarrhea, amebic dysentery, giardiasis, pinworm, roundworm, hookworm, and tapeworm.

They ascribe the transmission of trachoma, conjunctivitis, and yaws to rasping flies. Biting flies are said to transmit anthrax and tularemia. Other fly-transmitted diseases named are African sleeping sickness, leishmaniasis, onchocerciasis, loaiasis, bartonellosis, and sand-fly fever.

Horsfall in 1962 says in regard to the common housefly, *Musca domestica*, "Its exact role in the etiology of human diseases may never be fully known and undoubtedly varies according to the relative incidence of the flies. Situations where enteric diseases are endemic usually have sanitary conditions which also permit extensive development. Once the sanitation is good, incidence of both disease and flies declines."[120, p. 150]

Metcalf and Flint[86] in 1962 discuss (among other destructive insects) fruit flies as a source of damage or destruction of fruit (p.814). They refer to horseflies as the known carriers of loa loa, tularemia, and anthrax (p. 1008). They refer further to losses of human and supplies through the attack of these flies on meat and milk animals, whereby the animals lose weight and the yield of milk is decreased or the animals may be killed — either as a result of overwhelming attack or as a result of introduction of disease organism.

A nice distinction between the problems presented by solid wastes in the bulk and in small, isolated quantities is made evident by the horn fly. This fly feeds on cattle, but deposits its eggs only in fresh cattle droppings.[86, p. 954 ff] It does not breed in accumulated fecal wastes. Due to the damage it inflicts on major sources of human protein, its depredations can lead to human malnutrition (see also reference 37). Yet, unless attendants go about collecting freshly dropped dung, control of this fly through solid waste management has no meaning for the prevention of disease — either animal or human. Nevertheless, fresh animal droppings are solid wastes and must be classified as such; at least some attention to any health problems they might present is more likely to result from such an approach.

Of the housefly, Metcalf and Flint (p. 1031 ff) say: "This fly is naturally infected with the pathogens of more than twenty human diseases and many authorities believe that the fly is an important vector of typhoid fever, epidemic or summer diarrhea, amebic and bacillary dysentery, cholera, poliomyelitis, and various parasitic worms. However, adequate epidemiological evidence is available only for bicillary dysentery." They state that houseflies also serve as intermediate hosts of round worms of horses and tapeworms of chickens.

Tarshis (1962), in discussing infectious hepatitis,[530] quotes other authors on the transmission of this disease by flies.

In 1961, Bruch and others[551] had this to say about flies and diarrheal disease in Central America: "In Guatemala, the diarrheas and the dysenteries occur so frequently among children in the first few years of life that they are the leading cause of death for the population as a whole. . . . Environmental conditions clearly are responsible for many secondary sources of infection and for indirect transmission by vehicles and vectors. Pollution of water by human wastes occurs readily. Fruit is susceptible to contamination in its preparation and preservation. Flies are a hazard. On the other hand, living conditions are such as to favor direct contact spread. . . . Flies are especially frequent in September just as the rains end. The rather common increase in diarrhea in that month or in October may well be associated with flies."

In 1963, Greenberg and others[544] reported the results of a survey of a distribution of salmonellae in the fly population of a slaughterhouse in a central Mexican city. "Flies captured on offal had somewhat higher infection rates than those taken on manure: Ten types of salmonellae were recovered from the former and five from the latter [media]. In all, a total of twelve types was recovered from flies . . . *Salmonella derbi* was most prevalent, followed by *S. anatum* and *S. new brunswick*." The authors noted that the "slaughterhouse may serve as a meeting ground for the indigenous fly . . . and enteropathogenic bacteria from diverse geographic regions." They further stated: "This study has shown that flies can be superior indicators of the presence of salmonellae in such an environment. Fly pools have many more *Salmonella* types and higher percentages of positives than either rats or livestock. . . . Flies were infrequently found in the slaughtering rooms and were probably of little significance here as compared with [other] factors. The most obvious source of salmonellae for flies was carrion derived from the livestock. The higher recovery rates of salmonellae from flies captured over carrion compared with their counterparts from manure suggest that carrion is a better source of salmonella than manure is."[544]

The predominate type of salmonellae found in rats (namely, *S. typhimurium*), was not found in any of the positive fly pools. Greenberg, *et al.* stated: "Flies were not attracted to rat feces because of the superior attractivity and quantity of offal manure." The authors noted that the food preferences and dispersal patterns of the various flies helped to determine their vector potential. Certain fly species were noted to have little contact with man, whereas others, such as *Musca domestica*, were implicated in the fly–food–human cycle. Comment in regard to flies in relation to disease is of interest: ". . . in the abbatoir we studied, there is continuous year-round fly breeding, a fairly consistent feature of regions with high enteric disease rates, serving to maintain a constant vector pressure from flies. The public health danger from such mass-breeding, contamination, and dispersion of flies is clear. In the light of our findings, any program aimed at reducing diarrhea and enteric diseases, in general, must include measures to eliminate fly and rat populations from the slaughterhouse area."[544]

Gupta and Preobragenski in 1964, reporting on the epidemiology of trachoma in India, say this about flies: "It has been established that an important role is played by the common house fly. . . . the fly index rises before the onset of the two peaks of seasonal epidemic conjunctivitis. It has also been noted that the age-groups of children 2–4 years — which is most exposed to flies — [having] not sufficient sense and capability to keep the flies off their faces — gets the maximum [trachoma] infection" (p. 47).[681]

In 1964, Greenberg[543] made an interesting historical comment: "According to Aldronvandi writing in 1602, the Greeks and Romans were well aware of the possible spread of dysentery by the ingestion of flies accidentally falling into food." With regard to his own experiments, the author attempted to infect humans, without causing manifest disease, by using very small doses of *Salmonella typhimurium.* He states that the absence of response was probably due to the subthreshold dose of 10^3 and 10^5 organisms, at least 100 times lower than the number needed to produce symptomatic salmonellosis in adults. Greenberg concludes that "direct fly transmission of enteric infection to human adults appears doubtful where generally high infective doses are required. On the basis of this preliminary study, it seems likely that bacterial infections can occur through fly contamination of food. Flies have manifold opportunities to serve as vectors where poverty maintains a close association between domestic animals and people augmented by exposure of foods without refrigeration in market place and home. Fly mobility, demonstrated by the rapid dispersion of contaminated slaughterhouse flies, to market place, dairies, residential sites, and a neighboring town three miles away, poses a health problem for the entire community, transcending economic class and location."

Following his Mexican slaughterhouse study mentioned above, Greenberg, in association with Bornstein,[78] traced the flies they had demonstrated to be contaminated by Salmonella organisms to residential sites, market place, dairy, and a neighboring village up to three miles from their origin. He concludes that this dispersal constitutes a health hazard for the 100,000 inhabitants of the area.

Sacca[77] made some interesting quotations from sources not accessible to this study at that time. He states: "The fly's ability to transmit tuberculosis, leprosy, yaws, eye infections, et cetera has been proved. The medical importance of this species is particularly evident for the epidemics of trachoma: It has been shown that fly control alone is more useful than the administration of anti-biotics and sulphonamides to the human population." Brooke[553] considers that it is possible that flies may be involved in the transmission of amebiasis in rural areas where there is considerable contamination of the environment. He does not believe, however, that the fly is of particular importance in the spread of this disease.

In 1965, Moore, de la Cruze, and Vargas-Mendes conducted diarrheal disease studies in Costa Rica.[538] They state: "Variations in fly counts or in the bacteriologic quality of meat or milk, as determined, did not appear to be related to the variations of diarrhea morbidity." Domestic animals were observed to harbor enteropathogenic bacteria on some occasions but they were not demonstrated to be related to disease episodes in their owners. The authors felt that a bathing facility was needed to obtain the best effect from piped water, with which 94 percent of the homes were supplied.

In 1966, Shaker and others[691, p. 1586] reported finding a definite relation between infantile diarrhea and fly population in Kuwait.

DISCUSSION

It was recognized as early as 1913 that much data — fly-control experiments, improved bacteriologic techniques, and a better understanding of the epidemiology of the diseases in which the fly was implicated as a vector — were necessary to demonstrate conclusively that flies were indeed transmitters of disease. It was not until modern pesticides were available after World War II that fly-control studies were feasible, although, by hindsight, it appears almost certain that the conditions present in the Boer, Spanish-American, and American Civil Wars permitted the dissemination of typhoid fever by flies. The conclusion is weakened, of course, by the presence of other avenues of infection, but the evidence was and is convincing, especially following the experiments of Watt and Lindsay.[38] The fall in incidence of morbidity and mortality due to shigellosis in the populations they studied, following reduction in fly populations and their return to former levels with discontinuing control or development of fly resistance, seems to be quite similar to the reductions seen in typhoid fever when fly populations were reduced by natural causes. When one considers the comparatively small doses of typhoid organisms needed to infect susceptible individuals, the free access of flies to excreta and food in conditions of primitive sanitation, the positive association of the fly with typhoid fever in such conditions is difficult to question now that the relation to shigellosis is demonstrated. Nevertheless, the unknowns of disease-agent ecology in the climatic changes associated with fly destruction and suppression of breeding have not been resolved, and so there remains the possibility that the supposed relationship is coincidental.

On the other hand, there is no doubt that flies are mechanical carriers of a large number of agents pathogenic to man. There is also a great weight of circumstantial evidence (and a lesser amount of controlled research) relating to certain fly species that are contaminated by, or bred in, solid wastes with the actual transmission of human enteric and eye diseases, although infection may involve a number of factors other than that of waste itself. Of course, distinction must be made between those species that breed in, or feed from, wastes of human or animal origin and those that do not.

It is obvious that there is no simple relationship of flies to waste, but that species adaptability and adjustment vary greatly and must be considered in assessing an association.[689] Nevertheless, there is sufficient evidence of the linkage to condemn practices in the disposal of wastes which permit fly propagation.

Due to present United States agricultural and food-processing practices, fruit and vegetable residues can overshadow animals' feces as sources of fly production. If the resulting flies have access to human and animal wastes containing pathogens, a threat of disease transmission exists. The marked adaptability of domestic fly species, however, to many kinds of solid wastes for breeding or food supply does not permit oversight of any waste supportive of fly populations.

The systematic study of fly/waste/disease relationships has been neglected. The extent to which it must be conducted in respect to the solution of waste management problems is probably a function of the economics and sociology of fly-control in general. That is, the decision to support this kind of investigation will no doubt be determined by the public's *a priori* assessment of the risk — a typical paradox in public health — and therefore the degree of pressure exerted by the public, or the priorities established from a systematic survey of all waste management requirements within the entire environmental health field.

CONCLUSIONS

Domestic and some other species of flies are definitely established as transmitters of disease. They are bred in enormous numbers in many types of solid wastes. However, in only a small number of investigations[176, 33, 92, 76, 75] have there been any results from which a quantitative estimate of their part in disease causation was even remotely possible. It is highly probable that, in the presence of exposed human and animal feces, flies contribute significantly to disease and mortality. This is particularly true among infants and children; in some areas of the world, flies transmit typhoid fever and cholera, and on occasion cause high death rates at all age levels. However, much remains to be learned as to the contributions of numerous factors, such as the relation of seasonal changes on disease agents and vectors and their individual or mutual effect on disease incidence. In areas in which sanitation (safe water supplies, indoor washing and bathing facilities, waste disposal, and refrigeration) and personal hygiene are at a high level, flies have little opportunity to transmit disease.

The role of flies in disasters which interfere with sanitation in developed areas can only be surmised, but it could be a dangerous one in view of the probable low level of immunity to many enteric pathogens among the population in the United States (see references 739, 740, 741).

Although much research is needed to clarify the role of flies even in those few diseases in which flies associated with solid wastes have been determined to be transmitters, the evidence overwhelmingly demonstrates that control of solid waste against the breeding of domestic flies greatly limits their population. Present knowledge of the dynamics of disease transmission, exposure, dosage, etc., permits the conclusion that such limitation can contribute to the prevention of fly-borne disease.

RECOMMENDATIONS

The control or possible elimination of fly species associated with solid wastes and human diseases requires expanded application of known methods of waste control and research to increase knowledge of control. In the literature it was pointed out that fly control is associated with reduction of morbidity and mortality from some infectious diseases. However, it was noted that, even in developed countries, people are relatively tolerant of flies. In this country, carelessness in regard to domestic garbage[150, 391] and the feces of domestic pets[84, 391] is especially prevalent, and leads to significant fly production.

From these observations it is possible to outline programs designed to help control fly populations implicated in disease transmission areas in which research could be undertaken include the following:

Fly ecology
Fly predators and other enemies or parasites
Fly attractants and repellants
Interference with reproduction of flies
Species-specific pesticides
Physical agents of destruction
Waste control (control of breeding media)
Waste disposal (contamination control)
Public motivation toward fly control (including educational material, effective means of presentation and the like)

It should be noted that problems of implementation are not taken into account at this stage.

Of the items listed above, the last five are of most immediate concern to solid waste in its varied forms. There is a need to keep wastes apart from flies, to destroy flies finding access to waste, or to destroy the immature stages or eggs already in waste.

Another approach would involve implementing the following programs:

Known sanitary method of waste control and disposal
Expanded public education
Installation of full sanitary facilities in all homes throughout the country (eliminating privies in favor of flush toilets, providing piped water of sanitary quality, instituting pollution control, and providing indoor washing and bathing facilities)

All of these projects are of direct concern to solid waste management.

A solid waste/fly-borne disease relationship has been established to a degree sufficient to prove the need for remedial measures in waste management when flies have access to breeding media and fecal matter or other sources of pathogens. While there needs to be more research on the actual role of flies and the degree understood to which they contribute to any disease, this knowledge is not material to establishing the need to control waste in relation to the fly hazard. Such a need has already been firmly established.

Diseases Associated with Human Fecal Waste

GENERAL

1) Bacterial Infections. Typhoid fever, paratyphoid fevers A and B, cholera, and bacillary dysentery (shigellosis) are the enteric bacillary diseases in which man is the reservoir host (pp. 258, 260, 172, 378).[324] Typhoid is apparently an obligate parasite of man.[167, p. 864] Paratyphoid C is thought to be primarily a disease of animals.[324, p. 264] Paratyphoid B is sometimes excreted by animals.[324, p. 260]

The role of other bacterial agents such as *Escherichia coli* is not entirely clear, although they are implicated in diarrheal diseases of children, the malnourished, and the aged.

It is probable that the only important route for infection by these diseases is ano–oral, by either direct or indirect modes of transfer. Hand-to-mouth spread, directly or through food contamination, is probably the common mode of infection in this country,[332, 536, 534, 537, 538, 541, 542, 549, 550] although waterborne dissemination occurs.[40, 53] However, waterborne bacterial enteric infection due to municipal water-supply sources in the United States is uncommon[31, 143, 168]; nevertheless, even in advanced countries, waterborne contamination by bacteria from human fecal waste is common.[80, 103, 109, 586, 480, 584, 62]

Transmission of these diseases by flies is considered elsewhere in this report.

2) Viral Infections. Clarke and others (1962) stated that in the preceding 15 years, 70 new enteric viruses had been demonstrated in human feces and that the hazard of their waterborne transmission would increase as population growth caused greater contamination of water supplies.[755]

The viruses of poliomyelitis, of Coxsackie infection, and of infectious hepatitis are excreted in the stools of infected humans, but the modes of transmission are still being defined.[697, p. 8] Fecal transfer of polio virus, however, direct or indirect, is claimed by some to be the chief mode of infection for poliomyelitis[517, p. 32] and for Coxsackie disease.[417, p. 35; 697, pp. 8-9] Fecal transmission of infectious hepatitis is attested to by outbreaks of waterborne disease,[529, 552, 554] although person-to-person contact has been the chief means of spread in some epidemics.[528, 527] Chang[31] believes that outbreaks from municipally treated water supplies are unlikely. Weibel and others[40] are of the same opinion.

3) Protozoal Infections. Although intestinal protozoa of several species are found in man, only one, *Entamoeba histolytica*, appears to be a significant pathogen in the United States, unless other diseases, aging, or malnutrition exist.

4) Helminthiasis. The major worm infestations of human fecal origin are those due to the cestodes or tapeworms, such as *Diphyllobothrium latum* (fish tapeworm), *Taenia saginata* (beef tapeworm), *Taenia solium* (pork tapeworm), and those due to certain nematodes such as the pinworm *(Enterobius vermicularis)*. Also in this category are the human roundworm *(Ascaris lumbriocoides)*, the whipworm *(Trichuris trichiura)*, the human hookworms *(Necator americanus* and *Ancylostoma duodenale)*, and certain schistosomes.

All of these diseases depend on the transfer of the disease agent from human feces directly or indirectly to a susceptible human for new infection to appear.

Gordon, quoting the 1959 World Health Organization *Epidemiological and Vital Statistics Report*, (volume 12, p. 124), says that diarrheal diseases still rank among the five principal causes of death of young children in the United States. He states also that there is no guarantee that natural disaster or war will spare the United States, and that these catasthophes are and have always been associated with devastating intestinal disease.[552, p. 360; 548, 537, p. 353]

POSTULATION

The known or postulated paths of infection for fecal-borne diseases in which man is the reservoir can be shown as illustrated in Figure 4. This diagram emphasizes the diverse pathways involved. Four major routes are easily identified: vector-borne; soil-borne; direct contact; waterborne.

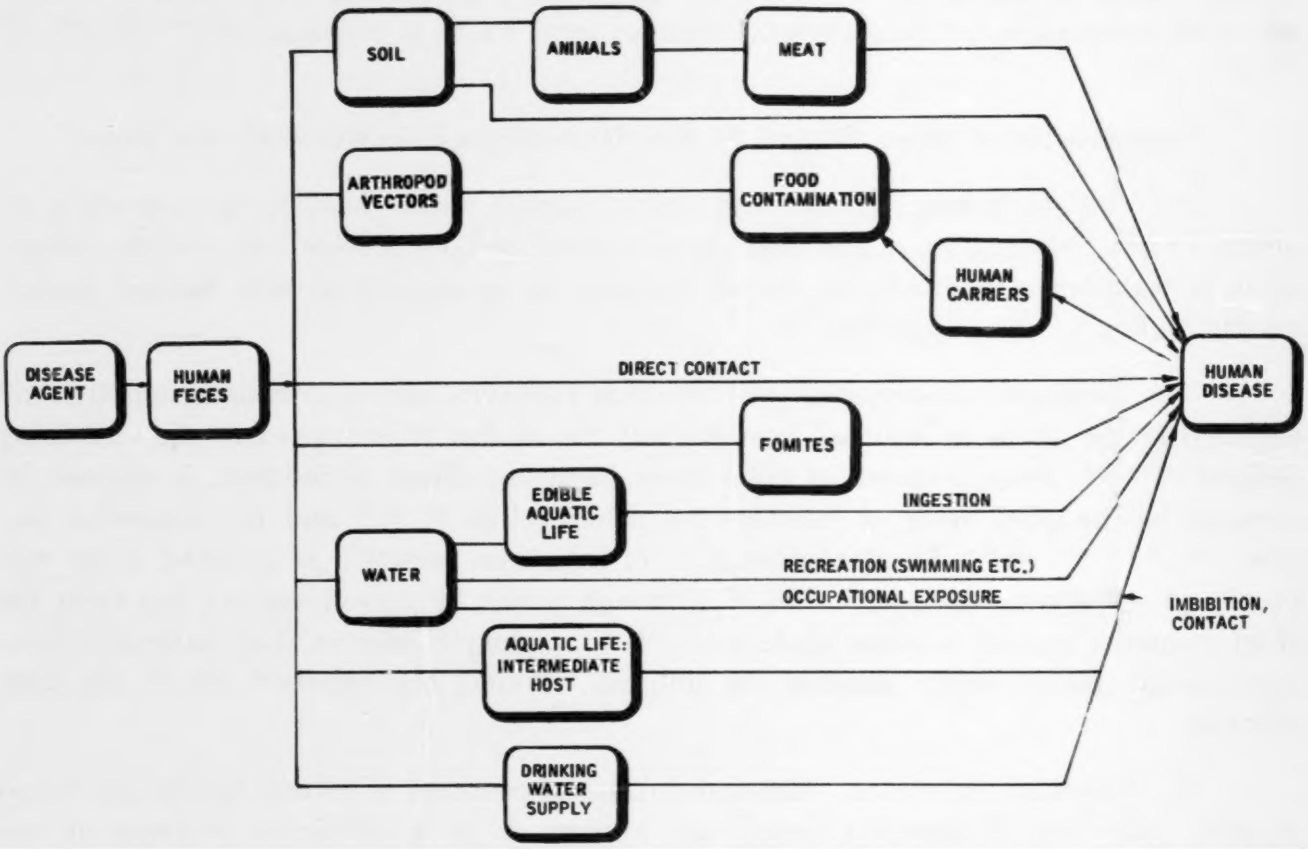
SOIL
ANIMALS
MEAT
ARTHROPOD VECTORS
FOOD CONTAMINATION
HUMAN CARRIERS
DISEASE AGENT
HUMAN FECES
DIRECT CONTACT
HUMAN DISEASE
FOMITES
EDIBLE AQUATIC LIFE
INGESTION
WATER
RECREATION (SWIMMING ETC.)
OCCUPATIONAL EXPOSURE
IMBIBITION, CONTACT
AQUATIC LIFE: INTERMEDIATE HOST
DRINKING WATER SUPPLY

FIGURE 4. Human fecal waste/human disease pathways (postulated).

One pathway is of special interest in that two biological agents potentially associated with waste (that is, the disease agent itself, and a vector — the fly) are implicated. Because of its still incompletely understood role and its possible importance to communicable disease, fly-borne disease will be considered as a special case.

EVIDENCE (SPECIFIC DISEASES)

1) Amebic Dysentery and Other Intestinal Protozoal Infections. Alone among the amebas, *Entamoeba histolytica* is known to invade the intestinal wall of man. In

1935, Craig[318] discussed the transmission of amebiasis through the use of night soil on gardens (pp. 46-47), the importance of flies as transmitters of the disease agent (pp. 47-48), and an epidemic of the disease observed by him and believed by him to be vectored by flies (pp. 56-58). This latter observation he supported by various epidemiological arguments.

In 1939, Manson-Bahr[321] reviewed the epidemiology of amebiasis and quoted the conclusion of Thomson and Thomson that houseflies *(Musca domestica)* could transport cysts and pass them unchanged in their dejecta. Manson-Bahr concluded that, under certain conditions, houseflies could act to disseminate amebic dysentery to a "somewhat limited extent." He also felt that transmission was possible by human excreta through the agency of vegetables and fruit grown where human "nightsoil" was used as fertilizer.

Craig and Faust[319] stated in 1940 that "the transmission of *E. histolytica* from person to person is usually accomplished through the ingestion of food or drink contaminated with feces containing the cysts of this parasite." They further stated: "The usual methods of transmission of *E. histolytica* are by the contamination of food or drink with the cysts by a polluted water supply; through the handling of food by infected individuals; by the droppings of flies and other insects; and through the use of human excrement in the fertilization of vegetable gardens." They felt that fly-transmission was important in "military camps, work or recreation camps, and wherever large numbers of individuals are gathered together and flies are prevalent."

In 1940, Tsuchiya, [94] in a study of the incidence of intestinal protozoa among medical and dental students, reported that the prevalence of 2 to 5 percent found was approximately that discovered in New Orleans and Philadelphia in other studies. He stated that, with one possible exception, those students harboring *E. histolytica* gave no history of diarrhea or symptoms suggestive of clinical amebiasis. Tobie, [114] in 1940 used isolates from apparently healthy carriers among humans to infect all of a group of 26 dogs. Amebic lesions were verified in the dogs at necropsy.

Faust[113] in 1941 in necropsy examination of accident cases in New Orleans, found *E. histolytica* in 13 of 202 cases. Other "parasitic infections" were found, and 42 cases were positive for one or more parasites.

In 1942, Strong, [198] in reviewing amebiasis, referred to the fecal transmission of the disease and gave the mode of infection as (a) contamination of food or water by feces containing the cysts, (b) the droppings of flies or cockroaches, (c) human excreta used as fertilizer, and (d) polluted water supplies (p. 493). He quoted Lynch (1915) as finding *Entamoeba histolytica* in rats in the United States, and quoted Tsuchiya (1939) as indicating that the rat possibly infects man with this disease (p. 494).

In 1947, Roberts[185] implicated several genera of flies in the transmission of amebiasis. This author stated, however, that it is now generally believed that, in urban areas, polluted drinking water and food-handlers are the main sources of infection with amebic dysentery, quoting Craig (1935) and the United States Public Health Service (1936). This author also stated: "In rural areas, however, particularly in the tropics, flies may prove to be a greater importance . . . having readier access to infected excreta. . . ."

In 1949, Pipkin[27] concluded from his experiments with flies that the "external carriage of stages of *E. histolytica* probably plays no important role in the transmission of amebiasis except in cases of gross neglect of sanitary rules" and that "it seems safe to conclude that although not impossible under usual circumstances, the ingestion of trophozoites by flies and subsequent deposition in their vomitus on human food or drink is not of great epidemiological importance." The author did indicate, however, that the internal transmission of cysts of the ameba in the feces of the fly "offers a potential natural method of transmission under conditions commonly occurring in backward and rural sections, which might possibly explain community amebic infections."

In 1957, Neal[332, p. 31] made the following statement: "Information concerning the dispersal of parasitic amoebae is almost entirely confined to the pathogenic species *(Entamoeba histolytica*, which infects man." Hunter and others, in 1960, referred to fecal transmission of this disease.[517, p. 271] These authors state that "while man is the principal reservoir of infection, amoeba which are morphologically similar to *E. histolytica* have been recovered from the dog, cat, rat and pig and various types of monkeys." Transmission of the infection from one individual to another is said to be accomplished by a variety of mechanisms. The housefly and cockroach are implicated, and it is said that in some areas flies probably are important in the spread of amebiasis. Another mode of transmission is the fecal contamination of water, either through surface streams, shallow wells, or springs. These authors also refer to the use of human excreta as garden fertilizer, and transmission by means of contaminated vegetables and fruit.

Beye and others,[575] in 1961 found a 3.6 percent prevalence of *E. histolytica* in Puerto Rican preschool children born in Puerto Rico but living in Philadelphia. It was their belief that the overall intestinal parasite reservoir among domestic and foreign agricultural migrants is large.

Chang[31] in 1961 refers to the fact that only four waterborne outbreaks of amebiasis had been recorded in the literature to that date. He stated that "the fact that the ratio of cyst density to that of coliform organisms in sewage (estimated on the basis of a 10 percent carrier rate) is approximately 1:100,000 and that cysts tend to settle out in sewage, sewage effluent, and water may very well explain the unlikelihood of water-borne amebiasis associated with municipal water supplies using even heavily polluted water as their raw source." He went on to say that all outbreaks had been traced to sanitary defects in the plumbing system, but the editor noted that involvement of organisms in public water supplies was still "in most cases . . . a matter of speculation."

In 1961, in the *Proceedings of the American Society of Civil Engineers*,[12] the following statement is made: "The resistance of the spores of *Entamoeba histolytica* to chlorine was so great that the absence of coliforms was no guarantee that the cysts were also absent. . . . where chlorination was the sole method of water treatment, the protection against amebic dysentery was nil."

Scott and Littig[395] in 1962 referred to the transmission by domestic flies of amebic dysentery and the protozoan disease giardiasis.

In 1962, Hoare[555] said: "Although [amoebiasis] is a typical anthroponosis, . . . natural infections with *E. histolytica* have also been reported from rats and dogs. But, since there is some evidence that the infected animals had been in contact with human cases of amoebiasis, it is thought that they derived their infection from man. This view was supported by the fact that rats and dogs are highly susceptible to infection with human strains of the dysentery amoeba" (see also reference 310).

In 1964, Brooke[553] reported that, on the basis of special classification of *E. histolytica,* it was estimated that the rate of infection for the general population in the United States was probably below 5 percent. However, he noted that the prevalence was higher in Southern states, rural areas of the country, lower socioeconomic groups, patients in mental hospitals, and travelers from tropical countries. He quoted Dr. Elsdon-Dew as stating that there may be very little correlation between the prevalence of the parasite and the occurrence of amebiasis.

Brooke also said that a survey of four selected areas in and around Little Rock, Arkansas, revealed 3.3 percent infection with *E. histolytica.* He stated that amebiasis will be found wherever poor sanitation exists, and noted that many of the reported epidemics have been in the temperate areas of the world. He quoted a survey by the Communicable Disease Center on five Indian reservations in which ranges in prevalence were from 1.5 to 33 percent. The survey did not permit correlation between any particular environmental, social, or economic factor and the level of prevalence. He noted prevalence of up to 70 percent infection with *E. histolytica* in wards of mental institutions. He felt that the transmission of the parasite in rural areas is probably through direct contacts with the fecal-contaminated environment. He stated: "The primitive sewage disposal, surface or pit privies, or no facilities at all is undoubtedly responsible for the contamination of the rural environment. In addition to the direct means of spread in the rural communities, consideration must be given to the possibility that contaminated wells, flies, and food-handlers may be involved in the transmission." He reported that for a nine-member family in South Bend, there were eight *E. histolytica* infections, adding that "upon investigation it was found that this particular family, unlike the others, was living in crowded quarters and had an outside privy and a private well."

In 1964, Dixon and McCabe[109] refer to the finding of *E. histolytica* cysts in sewage.

2) Cholera. In 1911, Herms[327] referred to the circumstantial evidence against the housefly in connection with cholera. He stated that cholera was one of the first diseases with which flies were associated as transmitters and "the experimental evidence that since has been produced is no less complete than in typhoid fever." He said also that "Without advancing the evidence as presented by Nuttall (1899), the following statement made by that eminent authority will serve the purpose, *viz.;* 'The body of evidence here presented as to the role of flies in the diffusion of cholera is, I believe, absolutely convincing.' " The data collected by Nuttall was not available in this study. In 1913, however, Graham-Smith [121] stated that he felt that further evidence was required to make the fly–cholera association.

In 1931, Gill and Lal [361] said in summary of their work: "The experiments recorded above are admittedly meagre, and it is not at present claimed that they justify the conclusion that a true host–parasite relationship exists between the fly and the vibrio. It would, however, seem in the first place that the vibrios are capable of surviving in the fly for over a period of at least five days. . . . About the fifth day . . . the fly is capable of infecting food by its faeces. . . . It has been shown that the infection of milk via the proboscis can take place up to 24 hours . . ." They also said that "it would be premature at present to discuss the significance of these observations, but they seem to suggest that . . . [the fly] may play a more important part in the transmission of cholera than has hitherto been suspected." These authors conclude that "cholera may perhaps have to be numbered among the insect-borne diseases" and that "it would moreover appear that in hot countries, insect transmission is of predominant importance and that the strikingly dramatic outbreaks associated with the massive pollution of water are relatively rare incidents if not accidents in the natural history of the diseases."

Gill and Lal also state that "the provision of piped water-supplies and the rigorous disinfection of the drinking water, combined with other routine measures, in the absence of measures designed to eliminate the gravely insanitary conditions which almost universally prevail in Indian towns have proved of little value." They conclude that "one of the most important, if not the most important, method of controlling cholera is the provision of an efficient sanitary control, more especially in connection with the collection, removal and disposal of night-soil and refuse." In 1939, however, Manson-Bahr [321] stated that he considered cholera to be mainly a water-borne disease.

In 1951, West [90] made the following statement: " . . . cholera . . . . etc., are listed as bacterial diseases with the implication (but no factual data of an epidemiological nature) that they are transmitted to human beings successfully by flies."

In 1952, Coffey and Dunn, [465] without providing data, implicate the fly as a transmitter of cholera.

In the report of a World Health Organization expert committee, [232] the following statement is made: "Waterborne diseases take a major toll and overshadow other environmental health needs in importance; the enteric diseases, including cholera . . . are the leading causes of death and disability in areas occupied by more than two-thirds of the world population." The report further states ". . . the safe collection and disposal [of solid wastes] is largely a problem of administrative willingness, of finance and of priorities. The problem of night-soil, however, is an exception for which further research is required. Research should continue in developed countries into improved methods of disposal." Another World Health Organization report[234] in 1965, states: "In one large metropolitan area in the tropics, a focus of endemic cholera has maintained itself for decades; it is believed that until adequate water supply and sewerage facilities are provided, this focus will continue to threaten other areas of the world with epidemics of this serious water-borne disease." The report goes on to say, "The Committee wishes to draw attention to the need for organizing the transport of garbage in the most sanitary and efficient way possible (especially in tropical countries), in order to assure proper control of diseases such as typhoid, plague, dysentery, cholera, etc."

3) Coxsackie Disease. In 1954, Melnick and others[28] recovered virus from flies in residential areas "where no source of contamination was obvious". There was no evidence of multiplication of virus within the flies, but it was found to remain in them or their feces for up to two weeks. These investigators stated that the recovery of the Coxsackie viruses was more regularly accomplished from sewage than it was from flies. They state that such virus had been found in the influent as well as effluent flow at sewage disposal plants. However, at times, it apparently did not survive, in detectable amounts, the trip from residential areas to the disposal plant.

In 1957, Smith[332], quoting a 1954 report by Melnick, Emmons, Coffey, and Schoof, stated that there was no evidence that infected flies played any part in the epidemiology of Coxsackie virus infection.

Kelly and Sanderson[18] reported in 1960, on isolating Coxsackie virus from sewage, both raw and treated. Chang in 1961 described the fecal origin of 30 types of Coxsackie virus, and expressed some amazement at the "complete absence of water-borne outbreaks of Coxsackie . . . virus infections . . .".[31]

Clarke and others,[21] in 1961, referred to the failure of activated sludge treatment of sewage to remove Coxsackie virus if the effluents were unchlorinated. Clarke stated that activated sludge would remove 90 percent or more of the enteric viruses added to sewage, but that disinfection was necessary if a virus-free effluent were to be obtained. On the possible infectiveness of sludge, he stated that only a very small amount of virus could be recovered indicating that the sludge–virus complex is extremely stable or that most of the virus is in some manner inactivated.[21] A Committee of the American Society of Civil Engineers reported in 1961[12] on the need to disinfect fecal-contaminated water. In the face of Clarke's statement, it is interesting to see that Mack and others "noted a higher percent recovery of viruses [polio virus, Coxsackie, ECHO] from settled activated sludge samples and from the liquid phase of sewage samples . . ." This report also refers to a "wide variation in virus removal efficiency of the flocculation process as studied by several workers."

In a 1964 report by Lamb,[98] group B Coxsackie and polio viruses were said to be most commonly found in sewage and river water collected in the period of July to

November, 1962, in a metropolitan area. Fifty of the samples collected were found to contain virus. Dixon and McCabe, reporting in 1964, stated that "many enteric viruses (Coxsackie, for example) may be present in domestic sewage . . . " A report in 1964 by McLean[3] is of interest in that it, like many others, indicates the difficulty of attempting to control many diseases merely by attacking one phase of the total problem. He referred to the spread of vesicular exanthem caused by Coxsackie A16 virus in Toronto, in 1957 — an action which may have been aided by the use of backyard swimming pools. It was also of interest that despite fairly heavy contamination on several occasions of one Toronto Beach with sewage, viruses were not recovered from the polluted area.

4) Infectious Hepatitis. Fecal contamination of water supplies by the virus of infectious hepatitis is evident in almost all studies of the transmission of this disease.

In 1959, Mosley[344] commented on the probable water-transmission of infectious hepatitis and the fact that the virus is excreted in the feces, but was puzzled by the comparative rarity of waterborne epidemics of the disease. Hunter and others in 1960[517] referred to the experimental evidence that human feces "constitute a source of virus for natural infection and limited explosive water-borne, milk-borne, and food-borne epidemics . . ." (p. 54). These authors stated further that "epidemics are particularly prone to occur under conditions of poor sanitation and hygiene which favor the dissemination of human fecal material."

In 1961, Dauer[168] stated that the total number of hepatitis cases reported in the United States increased from about 23,500 in 1959 to about 40,000 in 1960. These cases included serum hepatitis infections as well as infectious hepatitis. Dauer reported that "Epidemiologic investigations indicated that three outbreaks could be classified as water-borne and one as food-borne. Two of the three water-borne outbreaks occurred among school children whose drinking water at their schools came from wells. In each instance, contamination of the well with sewage was demonstrated."

In 1961, Chang made the following statement, "It appears that water-borne outbreaks of infectious hepatitis resulting from the use of municipally treated water supplies are unlikely. These outbreaks may occur, however, if the raw source is so grossly polluted by domestic sewage that the concentration of viral agents is such that it cannot be reduced below infectious levels by the treatment procedures available to the local water system . . . The great majority of cases (reported in 1960) were sporadic and of undetermined origin. At the present, epidemicological evidence points to personal contact as the major route of transmission."

A Committee of the American Society of Civil Engineers reported in 1961,[12] that "complete control of the hepatitis virus . . . in drinking water depends almost entirely on the disinfectant, as the virus is not eliminated or inactivated by coagulation, settling and filtration."

In 1962, in a discussion of a talk by Woodward,[17] the comment was made, "Cities with filtered water supplies show higher average morbidity rates than do cities with ground water supplies. This suggests that hepatitis transmission may be through water."

In 1962 Kabler[13] stated: "Infectious hepatitis is currently the only viral disease of humans for which the water-borne route of infection has been generally accepted, although it is believed that person-to-person contact is the more frequent method of transmission."

In 1962 Tarshis[530] quoted MacCallum and Bradley to the effect that human volunteers were readily infected by human feces administered by the oral route. This author also referred to his own investigations regarding fecal-transmission of the disease through cockroaches.

In 1963 Langmuir[88] referred to the initial establishment of raw shellfish as "an occasional factor in the spread of infectious hepatitis . . . in this country."

In 1963 Maynard,[528] reporting an epidemic of infectious hepatitis, stated: "There was good evidence of transmission of infection through person-to-person contact and the epidemic curve suggested this is the principal mode of spread." Where water barrels were used as sources of water for homes, he stated that evidence of coliform contamination of samples taken from these barrels indicated "the very real possibility of faecal contamination of water and possible spread of virus through this medium within the homes." Mosley, in 1963, reported on outbreaks of hepatitis in urban areas and concluded that there was a definite relationship between the incidence of the disease and the type of sewage disposal available. " . . . It was demonstrated that the high rates were associated primarily with the presence of privies."[529] He referred further to a report by Eaton of the significance of municipal sewage disposal facilities in regard to a hepatitis epidemic in Canada. The attack rate in families without sewage facilities was almost four times as high as in those with municipal sewage facilities. However, the author commented further: "The association of a higher attack rate with the presence of privies does not indicate that the privy *per se* plays a direct role as a fomite in the transmission of the disease. Probably more important is the lack of running water for washing at the toilet." He stated further that the epidemiological patterns suggested that the intestinal–oral spread of the disease is the major route involved and that "close personal and social contact with a case seems to be required."[529]

In 1964, Dixon and McCabe[109] referred to the occurrence of infectious hepatitis among construction workers at a sewage treatment plant but gave no data to support a causative relationship, and stated "A search of the literature shows no extensive surveys attempting to determine the actual incidence of infectious hepatitis in sewage workers and treatment plant operators."

In 1964, Weibel and others referred to the transmission of hepatitis by means of drinking water.[40]

In 1964, McLean,[3] discussing contamination of water by viruses, said, "Epidemiologic evidence gathered during several . . . outbreaks in Pennsylvania, New York, Kentucky, Austria, India and Australia has strongly suggested that they were caused by drinking water contaminated with infectious hepatitis virus. In this he quoted J. W. Mosley in that fecal contamination of the water supply was noted in each of these studies.

In 1965, Lobel and Robinson[527] noted, both in review of the literature and on a report of an outbreak of infectious hepatitis, a definite correlation between poor hygiene and sanitation and the incidence of disease, especially among children.

In 1964, a World Health Organization Expert Committee on Hepatitis[699] referred to the successful experimental transmission of this disease through the use of fecal filtrates from infectious hepatitis patients to volunteers by the oral route. The report stated further that "available data indicate that most cases of infectious hepatitis are due to person-to-person transmission, the effectiveness of which appears to be related to the closeness of contact. Persons living in the same household as a patient are at the greatest risk . . . In most studies the lower socioeconomic groups have a higher prevalence of infectious hepatitis in childhood, presumably as a result of greater crowding, poor sanitation, and less adequate personal hygiene." Other means of transmission noted are waterborne and food-borne mechanisms, including municipal water supplies, milk, raw shellfish, and prepared foods. With regard to vectors, the report stated, "Mechanical transfer of the virus from faeces to food or eating-utensils by flies and cockroaches has been suggested, but there is no evidence that this mode of transmission has any significance."

In 1965, Burns[83] stated, "The epidemic indications point to transmission (of infectious hepatitis) by contact (oral or nasal?)." He refers to other work in which the disease "has frequently been transmitted by feeding fecal suspensions to volunteers . . . ."[83]

In 1965, a report of a World Health Organization Expert Committee[234] implicated fecal contamination of drinking water in the spread of infectious hepatitis.

5) Poliomyelitis. In the period 1912 to 1913, there were a series of experiments which, according to the experimenters, successfully established that the stable fly *(Stomoxys calcitrans)* was capable of transmitting poliomyelitis to experimental animals.[36, 369, 370, 189]

In 1939, Paul and Trask[233] stated that poliomyelitis virus could be readily isolated from the stools of human patients and noted their own experiments in which monkeys were shown to develop poliomyelitis following inoculation of material prepared from sewage. In 1941,[367] the same authors referred to other experiments associating the poliomyelitis virus with human feces and sewage. They stated that, in addition to person-to-person, "there may also be a variety of other channels in which contaminated food, milk and water, or conceivably insects, mammals, or birds, play a part. That water-courses, and particularly water courses polluted with sewage, may be related to one of these channels is suspected."

Sabin and Ward in 1942[366] demonstrated the presence of poliomyelitis virus in flies trapped during outbreaks of this disease. They were unable to discover the source of the fly-carried virus. The fly species implicated was *Musca domestica.* They also stated "that . . . epidemiologically poliomyelitis seems more to resemble diseases like typhoid fever and dysentery in which the chief reservoir of infection is in human excreta and both direct and insect spread may be possible."

The fly was implicated further in the possible transmission of polio by Trask and others[159] in 1943, in which the virus isolated from flies was used successfully to transmit the disease to monkeys. In 1949, Melnick[365] also successfully infected monkeys with virus recovered from flies.

In 1953, Francis and others[364] referred to earlier recovery of the virus from stool specimens taken from outdoor privies. In the current study, they reported the recovery of polio virus from privies in four small Texas towns and stated: "A marked correlation existed between the presence or absence of poliomyelitis virus isolations from privy specimens and from the flies tested in a corollary study by Melnick and Dow."

In 1957, Dick[332] stated: "Claims have been made that biting flies could transmit polio virus (quoting the 1912 studies), but McFarlan, Dick and Seddon — 1946 — and others have been unable to confirm this either experimentally or on epidemiological grounds." This author also referred to the material quoted above in regard to polio virus and flies. Dick stated further, "There is no evidence of multiplication of the virus in flies. Whether or not the virus is carried purely mechanically is uncertain. Although most laboratory studies suggest that internal contamination of flies is a possible mode of transmission of poliomyelitis, there is no evidence that it is of any epidemiological importance. The best field evidence in this respect comes from the work of Paffenbarger and Watt (1953) who clearly demonstrated that fly control instituted before and during an outbreak of poliomyelitis failed to reduce the number of cases or to affect the time course of an epidemic in fly-controlled cities."

In 1959, Mosley[344] stated: ". . . [a waterborne route] has been postulated for poliomyelitis, but no studies have been reported in which conclusive evidence was obtained."

In 1959, Moore[584] reporting on studies in Britain relative to seaside bathing; stated: "We have had very little success in isolating . . . pathogens [except bacteria] from sewage-polluted seawater, partly no doubt because of lack of suitable enrichment procedures. Attempts to isolate polio virus failed." This author went on to say, "Study of bathing histories of poliomyelitis patients strongly suggests that in poliomyelitis patients who have bathed in the weeks preceding the onset of illness, the history of bathing is probably irrelevant."

The 1960 *Manual of Tropical Medicine* by Hunter and others[517] has already been quoted in regard to the indirect and direct means of transmission of polio from feces.

In 1960, Kelly and Sanderson[18] reported the finding of polio virus in sewage.

In 1961, Herms[195] referred to the fact that flies can harbor polio virus and, under proper circumstances, can transmit it . . . However, proof is lacking." In support of this, the Texas studies of Watt and Lindsay were quoted.[38]

In 1961, Chang[31] stated, "Of all the enterovirus infections recorded, only two water-borne outbreaks have been reported, both of poliomyelitis."

In 1962, Wiley and others[131] sampled sewage for poliomyelitis virus during an epidemic and reported "Overall recovery rates in twelve sample series were: 59.6 percent for polio virus type I, 7.4 percent for ECHO viruses, and 3.2 percent for type III polio virus. Enterovirus isolations varied from zero to 100 percent by sampling area, corresponding with case rates in the various socioeconomic areas. The recovery rates for type I polio virus from sewage, varied from 37.5 to 75.0 percent in different weeks." Also in 1962, Kabler[13] referred to the water-borne outbreaks of polio reported by Clarke and Chang, but stated that poliomyelitis infections do not appear to be related to water transmission.

In 1959, Downey began a study "To define more clearly the association of enteroviruses and flies" and, in 1963, reported, "In an urban area . . . where fecal material is disposed of in the sewage system, it is likely that virus can be recovered from flies only in the presence of infection of epidemic or near epidemic proportions."[735] The author was successful in recovering polio virus type I strains regularly in areas with a high degree of polio virus infection as manifest by clinical disease. "The converse relationship was noted in the second summer of the study when no clinical disease due to enteroviruses occurred [in the study locale] and no enteroviruses were recovered from flies."[735]

In 1963, Horstmann[697] stated: "The exact manner in which polio viruses are transmitted from one person to another is imperfectly understood. Close association, however, such as exists in the family setting, is important in giving rise to contact infections . . . The main portal of exit is the intestinal tract and large quantities of virus can be found in the feces often for many weeks and occasionally for many months . . . Whether virus travels from the pharynx of one person to the oropharynx of another, or whether the fecal–oropharyngeal circuit is the major one, has not yet been firmly established . . . However, although the evidence is not conclusive one way or another there is perhaps more data to support the view that poliomyelitis is an enteric infection spread primarily by contaminated excreta. Thus a poor sanitary environment is conducive to its dissemination, a feature which does not have a parallel in infection spread by the respiratory route . . . Taking all the evidence together, the fecal–oral route seems the more important one, although direct pharyngeal-oral pharyngeal spread may also play a role, particularly in epidemic situations." With regard to other modes of spread of the disease, she said, "Extra-human sources of polio virus spread . . . have never been regarded as being of particular importance. Although many animal and arthropod hosts collected in nature have been tested over the years, only flies and cockroaches have yielded polio viruses . . . The role of flies in dissemination of polio viruses has not yet been clarified . . . It is obvious, however, that flies are not essential to dissemination of polio viruses, since epidemics have occurred in Arctic areas under climatic conditions which preclude the presence of these insects . . . The interpretation of the findings is difficult, however, and at this stage we are still unable to fit together the data in a satisfactory manner or to assess the importance of flies in the over-all epidemiological picture." The author further stated: "Why epidemics of enterovirus infection and disease have such a striking seasonal pattern remains unexplained." [697]

In 1964, Lamb found that sewage and river water samples contained polio viruses. "About one-fourth of the river water samples contained virus. Group B, Coxsackie and polio viruses were most common . . . Raw sewage yielded the highest frequency of positive samples (80%)." [98]

In 1964, Clarke and Kabler[39] referred to the isolation of polio viruses from feces of "both paralytic and nonparalytic poliomyelitis patients". They stated, "The fate of viruses removed by activated sludge is not clear. Kelly's data suggests that viruses may be inactivated by biological antagonists in the sludge complex, a suggestion substantiated by the isolation from sludge of at least four strains of bacteria with antiviral activity. The data of Clarke and others indicate that virus removal by activated sludge is an adsorption phenomenon. They were able to recover only a very small percentage of virus from the sludge-virus complex, indicating that the complex is very stable or that the virus is in some manner inactivated . . . there are still wide areas of ignorance in our knowledge of the effectiveness of modern sewage treatment process in removing or destroying viruses in sewage." [39]

Weibel *et al.*[40] reported in 1964 only one waterborne poliomyelitis outbreak in the period 1946 to 1960. Hedstrom in the same year[554] experimentally polluted oysters with polio virus and indicated that they might "function as passive carriers of the virus."

6) Shigellosis. In 1942, Watt *et al.*[534] found a high rate (80%) of convalescent carriers of Shigella organisms. The investigators concluded, "Individuals recovered from diarrheal disease may continue to disseminate the infection days, weeks or even months." These authors noted a marked variability in carrier rates, however, among various locales in the United States. They state that the chronic carrier is uncommon and "of little importance to the spread of *Shigella dysenteriae*." [534]

In 1945, Hardy and Watt[533] studied acute diarrheal diseases in four widely separated regions. They reported that "in all areas investigated, *Shigella paradysenteriae* group was found most commonly in these diseases. No other recognized pathogen was identified in a significant proportion of the cases studied." They found "for every known infection (manifest source) there are numerous unrecognized infections (hidden source)." They also found a very marked variation in the prevalence of the organism, varying from 0.1 percent in New York City to 100 percent in New Mexico, with 4 percent found in Puerto Rico and 3 percent in Georgia. Deaths in positive cases were limited to infants under two years of age. Case–fatality rates were widely variant, ranging from no deaths in Georgia to 15.5 percent in New Mexico.

In 1948, Watt and Lindsay[38] studied the morbidity and mortality rates from enteric infections in towns undergoing fly control measures with controls provided by towns not undergoing such controls. They concluded, "In the area of high morbidity studied, a significant reduction in the amount of infection, disease, and death resulted from the degree of control established. The effect on Shigella infections was greater than on infections with the Salmonella group of organisms."

In 1951, Hawley *et al.*[135] demonstrated multiplication of enteric bacteria, (*Escherichia, coli, Salmonella schottmulleri,* and *Shigella dysenteriae*) within flies fed these bacteria. There appeared to be a threshold below which the number of organisms fed did not result in multiplication. In 1952, Dauer[53] stated that "faulty methods of handling food and poor hygiene on the part of food handlers were frequent findings in investigations of outbreaks of food-borne disease, in which foods other than milk were the vehicles of infection, according to 1951 reports of such outbreaks." There was only one report of an outbreak of Shigella infection with indefinite evidence of transmission by food or water, while seven outbreaks were thought to be person-to-person infections.

In another fly-control study in 1953, Lindsay and others[76] found that during effective fly control in an area of moderate diarrheal disease morbidity, the prevalence rate of Shigella and the morbidity rate from diarrheal disease was significantly lowered.

Investigating high infant mortality rates from diarrhea and enteritis in the San Joaquin Valley, California, Watt *et al.*[539] found a prevalence rate of 3.0 percent of Shigella infection in the children of residents in labor camps and fringe areas of towns in the area studied. These rates were considerably higher than that in children living in housing projects with higher levels of sanitation. These authors noted, "Shigellosis has virtually disappeared from the communities of the United States with a uniformly high level of sanitation and housing. The relative influence of various factors has not been defined because improvements in individual housing, water supply, garbage and sewage disposal, higher standards of living, and better education are usually concurrent and maybe accompanied by, or the result of, community-wide changes."

In 1958, Schliessmann *et al.*[545] studied the relation of environmental factors to the occurrence of enteric diseases in mining camps in eastern Kentucky from June 1954 through June 1957. High morbidity rates from diarrheal disease were found, with Shigella isolation rates of preschool children ranging from 0.7 to 10 percent in individual areas. The highest rates occurred in the four-year age group, while in the most poorly sanitated areas, very early infection was found, with the highest prevalence rates in the two-year age group. It was found that shigellosis "probably was responsible for the majority of acute diarrheal disease experiences observed in poorly sanitated areas, but was not a primary cause in the most well-sanitated area." They could not implicate the fly in the transmission of shigellosis, but transmission of enteric pathogens by polluted water could readily have occurred since water sources were subject to possible fecal contamination. They reported, "There were, however, no instances in which water quality could be implicated in disease outbreaks or correlated with seasonal differences in morbidity rates or Shigella prevalence. Lowest rates of reported diarrheal disease, Shigella-positive cultures and

Ascaris-positive stools were recorded among study families served by complete community sanitary facilities . . . The highest levels of [these] three indexes were reported from populations living where community sanitary facilities were entirely lacking. Individuals living at homes provided with inside piped water and privy excreta disposal reported approximately twice the diarrhea, and twice the Shigella prevalence, and over three times the Ascaris infection rate experienced by individuals using inside piped water and flush toilets. For the population groups using privies, Ascaris infection rates and reported morbidity rates were one-third lower, and Shigella infections were 50 percent fewer, among those who had water inside the house than among those whose water source was outside. Where the water source was outside the dwelling unit, Shigella and Ascaris infection rates were comparable regardless of water source location or relation to the premises. There were trends, however, to indicate that lower rates of Shigella and Ascaris infection accompanied the existence of installed bathing fixtures. Desirability of installed hot water systems was also indicated . . . The results of this study strongly support the premise that incidents of acute infectious diarrheal disease may be reduced significantly through selective modification of specific environmental factors within communities without regard to etiological or sociological differences. It is concluded that specific environmental improvements, based on a knowledge of local deficiencies, will invariably effect significant reduction in enteric disease."[545]

Moore[584] reported in 1959 on pollution of seaside beaches, "A colleague isolated *Shigella sonnei* once from direct plates of sea-water at a time when an extensive outbreak of dysentery was occurring in a community from which the sewage contaminating the sea-water was derived." He stated: "We have had very little success in isolating [Shigella] from sewage-polluted sea-water, partly no doubt because of lack of suitable enrichment procedures."

In 1961 Dauer[168] stated: "All outbreaks of shigellosis reported in 1960 were caused by *Shigella sonnei.* One waterborne outbreak followed a breakdown in the water treatment plant of a town . . . Another outbreak was traced to the water from a spring in a picnic area. Another rather large outbreak was reported in which the epidemiologic evidence pointed to some food eaten in a school cafeteria. In another instance students who ate in a college dining hall became ill with diarrhea, which was confirmed as shigellosis by laboratory tests of specimens. *S. sonnei* was isolated from the stools of a cook who had gastroenteritis 5 days before the outbreak. In another outbreak, *S. sonnei* was also isolated from a person who prepared potato salad for a buffet supper in a home."[168]

Sabin[550] stated in 1963: ". . . the impression gained from recent reviews is that the *Shigella* bacteria are *presumably* the chief pathogenic agents of morbidity and mortality from diarrheal disease in populations living under conditions of poor sanitation and hygiene . . . My own analysis of the available data led me to conclude that, particularly in children under 2 years of age, the age group that is most important from the point of view of mortality, the *Shigellae* and other specific bacterial pathogens, while still important, may frequently constitute only a small proportion of the etiological agents." The author noted that the high death rate due to diarrheal disease in general throughout the world was in sharp contrast to the rates in this country, in reporting, "The extraordinary low level of diarheal deaths in infants achieved in the U.S.A. and Canada by 1955 apparently was associated more with other improvements in the standard of living than the mere provision of water and sanitary disposal of excreta in the homes already in existence in New York City between 1901 and 1920 . . . On the basis of present knowledge that infantile diarrheal mortality has multiple causes, among which direct transmission of human enteropathogenic bacteria and viruses by dirty hands, consumption of food that has served as a culture medium for billions of bacteria, and malnutrition are perhaps the most important, it is not surprising that it remains an important problem until very high standards of living are achieved in a population."[550] The author stated that malnutrition is of especial importance in the rates of infection and mortality of diarrheal in general.

Gordon *et al.*[537] reported in 1963 on the incidence of diarrheas and dysenteries throughout the world beginning in the year 1900. These authors stated that "Shigellosis or bacillary dysentery is the most common specific enteric infection among diarrheas of children. In countries with good nutrition, . . . this infection (has been shown) to account for ⅔ or more of cases . . . Shigella is less common in diarrheas of preindustrial countries where diarrheal disease is more prevalent." The role of weaning and malnutrition in underdeveloped areas is emphasized.

In 1963, Greenberg *et al.*[544] were unable to recover Shigella organisms from flies or livestock in a Mexican slaughterhouse.

In 1964, Nakamura *et al.*[126] found that Shigella organisms did not survive as long in natural untreated seawater as compared with autoclaved or filtered seawater. "There was a wide variation in the ability of different strains to survive in untreated sea-water . . . The Shigellae were quite resistant to the osmotic effects of high concentrations of sodium chloride. However, survival depended a great deal on the holding temperature."

Reporting in 1964 on diarrheal disease in a children's home, Rosenstein found that "while the majority of cases are not of specific bacterial origin, a large number of children with Shigella and Salmonella enteritis continued to be seen . . ."[571]

Guardiola-Rotger *et al.*[536] reported in 1964 on the incidence of enteric organisms in Puerto Rico villages. Both villages studied had "poor living and unsanitary conditions." The climatic conditions were quite different. "Bacterial enteropathogens were obtained from 31.0 and 19.0% of the cases studied at Manzanilla and Cialitos, respectively . . . The incidence of shigellosis was higher at Manzanilla (10.3%) than at Cialitos (3.7%). *Shigella sonnei* was isolated from 6 of the 7 cases of shigellosis at Cialitos, whereas at Manzanilla the *Shigella flexneri* group was more prevalent. Heavy infestations with other parasites were also found."

In 1965, Schneierson and Bottone[549] studied Shigella prevalence in an underprivileged community by culturing the stools of patients admitted to the local hospital. The findings indicated "a considerable prevalence of shigellosis in the community . . . Most of the patients were children, 79% being under 12 years, and 60.6% under 5 years of age.

However, a greater degree of infection may be prevalent among adults (in the community) than is indicated by these data. Adults do not seek medical care for themselves as readily as they do for their children, especially since the disease is mild and self-limited in nature."

7) Typhoid and Paratyphoid Fevers. Early observations established the fecal transmission of typhoid or "enteric" fever and associated the spread with various direct and indirect modes of infection.[388, 548, 355, 354, 356, 360, 336] It was noted that failure properly to dispose of fecal material and poor sanitation and crowding were associated with the rapid spread of typhoid fever. Flies were noted to be present in large numbers where "enteric" was found. These early observers could not dismiss as merely coincidental the disappearance of enteric fever with the killing off of flies by seasonal change.

The circumstances under which these early observations were made are no longer prevalent in the United States. According to Dauer in 1952,[53] there were slightly more than 2,100 cases of typhoid fever reported in 1951 in the country as a whole, with only three outbreaks of typhoid reported. In the period 1960 to 1964, 3,376 cases were reported and, in the year 1964, reported cases were 486.[351]

The hazard of typhoid and paratyphoid infection as a result of bathing in sewage-polluted seawater was reviewed by Moore.[584] This author criticized earlier studies (New York, 1930's) because of the lack of modern bacteriologic techniques at that time. Moore examined 859 samples of seawater and stated, "The numerical preponderance of paratyphoid strains greatly exaggerated their importance . . . The few isolations of typhoid bacilli may, on the other hand, underestimate the numbers usually present because of the greater technical difficulty of culturing this organism." Epidemiologic studies were conducted, and it was reported that "An intensive effort has been made during the past four years to keep track of enteric fever patients giving a history that pointed to an association with sea-bathing. The medical officers of health of about 80 coastal administrative districts kindly looked through their records of sporadic enteric fever notified in the previous five years to see if the paratyphoid morbidity in costal areas differed significantly from the national figures. No evidence of an increased paratyphoid incidence in seaside residence emerged from a scrutiny of these figures, and surprisingly few of the figures recorded have been in the age group associated with sea-bathing. As a result of these inquiries, only four cases of paratyphoid fever giving histories that pointed to sea-bathing infection have been detected in England and Wales during the past three years. It so happened that all four were associated with one or another of two beaches that had been intensively studied by members of the committee, and from which paratyphoid bacilli of the relevant phase types had been isolated beforehand. Both [beaches] were grossly . . . contaminated with crude sewage . . . One may then sum up the evidence of the risk of contracting enteric fever from sea-bathing by saying that less than one case a year of those that have come to our attention have given a history that suggested a sea-bathing infection."[584]

Dauer in 1961,[168] reported on four outbreaks of typhoid fever in 1960: " . . . one

. . . was clearly waterborne, one . . . probably was waterborne, and two . . . were foodborne. In the first outbreak, six users of well water developed the disease. Investigation demonstrated that the well was contaminated from seepage from a septic tank used by a known carrier. In the other small outbreak the two persons affected lived in crowded, unhygienic surroundings, and had used water from a well that was susceptible to contamination from a septic tank. A resident of the immediate area who used these sanitary facilities was a typhoid carrier."

In 1964, Weibel and others[40] reported that, in the 15-year period, 1946 to 1960, there were 39 reported outbreaks of waterborne typhoid fever consisting of 506 cases. Eight deaths resulted.

Hans Fey[324] stated in 1964 that typhoid fever occurs partly through contact between persons, but chiefly through pollution of water and food. He stated that primitive living conditions and insufficient waste disposal are major factors in epidemicity. He stated further that paratyphus A infections had the same mode of transmission as of typhoid fever. In paratyphoid B, man is considered the primary source of infection in the great majority of cases; however, the disease organism is occasionally excreted by animals and transmitted by means of food. The latter mechanism occurs but rarely in Europe. This author considers paratyphus C as a zoonosis in that the organism is found in meat animals rather than in man.

In the same monograph Lachowicz stated: "A very serious problem in the control of typhoid is sanitation, especially that of the water supply in the sewage disposal in the geographic regions where water sources are scare and people are forced to use surface water (for example, some middle Asian Republics of the U.S.S.R.)."[324]

In regard to the presence of endemic areas of importance in this age of rapid transportation, the following conclusion is drawn in the chapter "Les Salmonelloses en Afrique": "Among the distribution of serotypes of salmonellae in the countries of Africa cited in example, . . . *S. typhi* is the most frequent serotype in man."[324] Also in this reference, the effect of war on typhoid prevalence in Eastern Europe was noted: "Before World War II typhoid and paratyphoid incidence in the U.S.S.R. had decreased to a level of about 50 cases per 100,000 population as judged from figures available with respect to the Ukrainian S.S.R. in 1940. There was a great increase in typhoid and paratyphoid incidence during the war but it ceased soon, and not only the inter-war level but also a further decrease in the incidence has been achieved. It may be illustrated again by the typhoid morbidity rate per 100,000 population in the Ukrainian S.S.R. which amounted to 57 in 1947, to 18 in 1950, to 13 in 1953, and to 12.3 in 1956. A decrease trend like this, however, has not been observed in all the Republics. As a result the average typhoid morbidity rate for all the country in the mid-fifties constituted about half of that in the last inter-war years, probably about 25 per 100,000 population. The decrease trend ceased in the 1950's." A similar decline of typhoid and paratyphoid incidence was reported for Hungary and Romania but it was stated, "This is to a certain extent an exception among the Eastern European States as there has been only little change in its typhoid–paratyphoid morbidity rate during the last 40 years. If, however, typhoid and paratyphoid are considered separately, a decrease of the typhoid incidence and an increase in the paratyphoid is apparent."[324]

In the same monograph, the decrease in "enteric fever" in Canada from the beginning of the century was noted. It was pointed out that "with the improvement of sanitation and hygiene, the wider distribution of pure (treated) community water supplies and of pasteurized milk, the limited use of TAB vaccines, improved methods of bacteriological diagnosis and a concerted effort on the part of medical officers of health to search out 'carriers,' the enteric fevers . . . typhoid and paratyphoid . . . have been slowly but steadily decreasing . . ." It was noted that in 1962, there were only 268 cases of "typhoid and paratyphoid" for all of Canada or a rate of only about 1.5 per 100,000 population.[324]

Middelkamp[541] stated in 1965 that, "Typhoid fever is still one of the most common febrile illnesses encountered in infants and children in tropical and subtropical countries. The most significant factors that effected the decrease in prevalence of this disease in the United States were: (1) The use of methods that provide us with sanitary food, water, and milk supplies; (2) detection of carriers and prevention of their employment as food handlers; and (3) The administration of typhoid vaccine. This paper is a current appraisal of our experience with typhoid fever in pediatric patients . . . during the 15-year period 1950-1964. Over this 15-year period there is a decrease in the number of patients hospitalized with typhoid fever . . . Sixty-seven percent [were] under six years of age. Twenty-eight of these children lived in the city while twelve were from rural areas." The investigation of the cases showed that five *Salmonella typhi* carriers were found in a survey of the contacts of the patients. Two of the carriers were the patient's mother, one a grandmother, one an aunt, and one a neighbor. One child, an infant, died. One child remained a carrier for five months following his discharge from the hospital. This author quotes Ashcroft as stating that, "Protection by good vaccines may be partial . . . The immunized individual may be protected against a small number (of organisms), but such an immunity could be overcome when a large number . . . are ingested."[541]

In a 1965 report, Van Der Schaaf and Atteveld[80] stated, "Biological sewage treatment is applied on a large scale in the Netherlands. In most cases suspended and dissolved organic matter is removed satisfactorily, but from the point of view of a veterinary and medical bacteriologist, the removal of pathogenic bacteria is far from complete. This causes contamination of the water in nearly all the canals, lakes, rivers, and even small brooks." These authors make the following interesting comment: "Although typhoid officially does not occur in Utrecht, *S. typhi* can be cultivated from nearly every sample of effluent. Sometimes it was possible to isolate *S. typhi* for the first time from samples which had been stored during one month up to six weeks in a refrigerator. By that time quite a number of other kinds of salmonellae had already died off and consequently no longer hindered the isolation of *S. typhi.*"

8) Tuberculosis. Solid waste has been implicated in the spread of tuberculosis.[23] Experimental transfer to culture media of the bacilli by flies was reported in 1907 by Buchanan.[360] The relation of flies to waste has already been documented in this report. Graham-Smith[121] in 1913 noted that flies could carry tubercle bacilli but that "In considering this relation to infection in the human subject the influence of dose must be taken into consideration" (p. 179).

In 1965, Szulga and others[6] were able to isolate human tubercle bacilli from the milk of cows and from sewage used for fertilizing pastures. In the same year, Buczowska[30] found that sedimentation and biologic purification did not remove tubercle bacilli which had survived in the sewage during the flow to the treatment plant. This author reported that disinfection of hospital and sanitarium sewage by means of discontinuous chlorination in "small, uncontrolled treatment plants" proved ineffective. It was noted that contamination of the domestic sewage tested was derived from an area where the morbidity index of active tuberculosis among the population was 124/10,000. The danger from the use of sewage for the irrigation of forage-crop fields was noted. It was also stated, "Slaughterhouse wastes may be regarded as contaminated if they are derived from slaughtering cattle with a high percentage of tuberculosis infection."

9) Worm (Helminth) Infestations. Fecal-borne human helminthic infections are well recognized. In 1940, Craig and Faust[319] stated that "Sanitary disposal of human feces containing the viable eggs of these tapeworms *(Diphyllobothrium latum)* will protect communities . . . In all known tapeworm infections, except ocular sparganosis . . . the portal of entry is the mouth. Sanitary disposal of human excreta and those of domestic animals constitutes the more fundamental and more urgent public health problem." In regard to *Ascaris lumbricoides,* the authors stated: "Man is apparently infected only with eggs from human sources. Human infection is acquired by ingesting fully embryonated eggs which have been accidentally picked up from the soil polluted by the same or other human beings, or from food or drink contaminated by viable eggs. Young children are more commonly infected than adults and more commonly pollute the soil." Schistosomiasis also arises from improper disposal of human feces. Although intermediate hosts (snails) are involved in the transmission of this disease, their infection derives from water polluted by human feces. The above authors stated that "All of the snails involved are sewage-feeders."[319]

In 1956, Lindsay and Scudder[75] stated that "Without indicating the degree of importance, it may be said that flies constitute one mode of transmission for the agents of such diseases as . . . helminth infections." The importance of improper disposal of fecal waste is contained in a statement by Smith,[332] who wrote in 1957, ". . . It has been estimated that perhaps some 200,000,000 people may be infected (by shistosomiasis)." This author also referred to the modern practice of crowding farm animals together so that they become contaminated by their own feces, whereas, in nature, they tend to avoid such contamination.

In 1958, Schliessmann and others[545] reported on the high rates of helminthic infections among children in mining camp areas in the United States. They noted that the incidence of infection was related to the method of disposal of human feces.

Hunter and others, in 1960,[517] also relate diseases due to helminths to improper human fecal disposal (pp. 411, 412, 419, 420, 498, 503, 530, 547, 553, and 556).

In 1961, Chang[31] stated, "Municipal water supplies are not known to be involved in spreading infections by any nematode. But small rural supplies in certain endemic areas could carry hookworm or strongyloidal larvae washed into the water from infected soil. These supplies constitute a mode of transmission of minor importance as compared to the soil itself." This author also speculated on the possibility of nonpathogenic nematodes ingesting pathogenic bacteria and so protecting these bacteria from sewage treatment processes, so that they might survive and serve as sources of human infection. He noted, however, that "The chances (for this) are so small that the possibility must be considered as very remote."[31]

In 1961, Beye and others[575] reported on the parasite reservoir in the United States and attested to the high incidence of helminthic infestations among the families of migratory laborers and among residents coming from tropical and subtropical lands. The possibility for spread to others in the community was noted. "Migratory agricultural workers number 1.25 million individuals, approximately one-half nationals from outside the United States and one-half workers from the Southern States. A population of this size may present public health hazards with respect to protozoan and helminthic infestations and diseases." They go on to say that "Although there are as yet no documented instances of greatly increased transmission of protozoa and helminth associated with agricultural migration, this does not mean that increased transmission is not occurring. It could reasonably reflect that few are looking for signs of infection and the disease is not yet manifest."[575]

Jenkins[227] stated in 1961 that ". . . the possibility of worm diseases being spread by sewage sludge is considered." He said, "Digested sludge from Pretoria could suffer a 100 percent reduction in viable ova of Ascaris, . . . but sometimes only 85 to 95 percent inactivation resulted." This author also quoted another study to the effect that digesting sewage sludge for 20 days at 35° C was necessary to inactivate the eggs of *Taenia saginata*, the human beef tapeworm.

In 1962, Thomas W. M. Cameron[558] had this to say: "Taking the world as a whole, it was realized that parasitic worms had an importance comparable with the bacteria, the protozoa, the spirochetes, the rickettsiae, and the viruses; in fact, in everyday deterioration of health, as causes of chronic debility, they are of more importance to the world than the acute infections caused by the microbes." He went on to say that ". . . the common pinworm *Enterobius vermicularis* . . . infects nearly 50% of individuals in temperate climates, even when a relatively high standard of hygiene is maintained. The trichina worm . . . can also be extremely common, as it is in the United States . . . . Hookworms and bloodflukes can be included among the causes of the half-dozen most serious diseases of mankind. Ascarids are just as common although possibly less serious and are widespread." The authors repeatedly refer to the importance of fecal contamination as a result of improper disposal, poor personal hygiene or other practices which lead to contamination of food or other agents of transfer.[558]

In 1962, Chang and Kabler[101] reported that "... The possibility that the effluent-borne nematodes are carriers of human enteric pathogens is quite remote under normal conditions."

In 1962, it was reported[316] that "The life cycle of (the common tapeworms) is maintained in areas where there is unsanitary disposal of human feces and access of the domestic animals to feed or water that has been contaminated with the excreta of a human being who harbors the tapeworm . . . The importance of the problem in different countries varies with the extent to which sanitary disposal of human feces is practiced, and with the meat eating habits of the people." This book quoted Schwartz (1956) as saying that in the United States about 16,000 to 27,000 infected beef carcasses were found annually in abattoirs under Federal inspection, "However, to point up the importance of local conditions, Schwartz (1938) cited three examples of outbreaks of bovine cysticercosis with high rates of infection due to contamination of the feed and water with human excreta."[316]

The importance of fecal sanitation is indicated in a report (1964) by Guardiola-Rotger and others[536] who found 80 percent of children in a village in Puerto Rico infected with helminths.

Human fecal contamination of the soil has been found by Beaver (1964) to be responsible for occupational disease . . . creeping eruption . . . among workers who must crawl over infected soil.[557]

In 1965, Fair and Geyer[215] reported that "the utilization of sewage sludges is circumscribed by the hygienic hazards found. Pathogenic bacteria, viruses, protozoa, cysts, and worm eggs, can survive sewage treatment and be included in the sludge. There, they will persist for long times and cannot be fully destroyed by digestion or air-drying. Although the numbers of surviving organisms decrease appreciably in the normal course of events, only heat-dried sludge can be considered fully safe . . . Wet-sewage may be pumped on to land and plowed under . . . Sewage sludges may be discharged into water. Sea coast communities may transport . . . sludge to dumping grounds at sea. Wet-digested sludge may be discharged into large streams (more particularly in times of flood runoff) . . . or it may be pumped to deep lying and hydraulically active portions of tidal estuaries."

In 1966, Walters and Holcomb[383] stated, "Nematodes ingest enteric bacteria in certain sewage treatment processes and carry them out into the receiving streams. The absolute number of pathogens found would seem to be of secondary importance. If any enterics are ingested it seems logical to assume that enteric pathogens would be ingested in large numbers if the concentrations were very great in the sewage as would be the case in a local epidemic of a disease like typhoid fever."

DISCUSSION

1) Amebic Dysentery. Amebiasis due to *E. histolytica* is endemic in this country: The literature does not permit a clear explanation of how it is maintained, but reservoirs obviously exist in both large cities and rural communities. Carriers exist and presumably transmit the cysts by direct contact and by contamination of food or water.

If, as has been stated,[31] transmission by water from municipal treatment plants is improbable, infections where treated water is provided must derive from gross fecal contamination of food or direct contact.

These modes imply, where treated water is available, insanitary disposal of feces, failure to use the water supply for washing, or both.

Increasing levels of infection are associated with heavy fecal contamination of the immediate environment[553]; when this occurs, direct person-to-person or vector transmission is said to take place.

2) Cholera. Cholera is not found today in the United States. It is a fecal-borne disease in which fecal contamination of the environment and direct contact appear to be the chief modes of transmission. It is frequently referred to as a water-borne disease. Flies are implicated as transmitters where they have access to human excreta containing the organisms.

There are endemic foci in the world which provide potential sources of dissemination to countries now free of the disease.

3) Coxsackie Disease and Poliomyelitis. Although the viruses of these diseases are found in human feces and in flies having access to feces, the modes of transmission are still somewhat obscure and will probably remain so for some time. The widespread but incomplete use of polio vaccine serves to confuse epidemiologic investigation of poliomyelitis spread. The consensus is that both diseases are fecal-borne rather than conveyed by the respiratory route. The degree to which they are transmitted by direct or indirect means is unknown.

4) Infectious Hepatitis. Transmission of this disease is chiefly through direct contact or fecal contamination of water supplies, with the former responsible for the greatest number of cases. Since there is a higher morbidity rate reported from cities using filtered water as compared to those using groundwater, there is a question as to whether municipal and other water and sewage treatment is sufficient to remove or inactivate the causative agent. (This comment applies to other viruses as well.) The failure or lack of sewage treatment to inactivate the virus is borne out by the finding of outbreaks traced to shellfish growing in polluted stream estuaries. Higher rates of infection associated with the use of privies incriminates this means of fecal disposal.

5) Shigellosis. This disease is apparently of lesser virulence in the United States than in countries of the Far East. Prevalence is related to overall sanitation, with the lowest levels found where full sanitary facilities are available and the educational level is high. The fecal source of the infection is obvious, although the mode of transmission varies. Good personal hygiene, waterborne disposal of excrement, and indoor water supplies and bathing facilities are all necessary for reduction of infection.

6) Tuberculosis. In the United States, there appears to be no evidence of fecal transmisison of tuberculosis, although this mode of infection seems possible in other countries.

7) Typhoid and Paratyphoid Fevers. In the United States, the typhoid carrier is the chief source of infection, regardless of the exact mode of transmission from the carrier to the victim. Carrier-contamination of well water from privies or septic tanks is documented. Where flush toilets and adequate treatment of water supplies are not available, waterborne outbreak of typhoid is possible. That sewage treatment is not necessarily dependable in suppressing typhoid is evident from European studies. Infection of children by familial carrier contact is a significant mode of infection.

The incomplete protection from vaccines complicates the interdiction of transmission and the development of the chronic carrier state is common.

8) Worm (Helminth) Infestations. The fecal transmission of human helminthiasis is well documented. Where environmental contamination by feces is permitted, high infestation and prevalence rates persist. The hazard of failing to provide good sanitation for migratory workers, both to themselves and to the general public, is made evident by the high incidence of helminthic infection among these groups. Sewage sludges have been found to harbor the eggs of pathogenic helminths, raising the question of the safety of using unsterilized waste sludge as fertilizers or soil conditioners.

9) General Discussion. There are a number of unresolved questions about the fate of fecal pathogens in the environment. The fact that typhoid organisms can be cultured from refrigerated surface water specimens in which suppression of other organisms has occurred, but not in unrefrigerated specimens, and the extremely low numbers of typhoid bacteria necessary for infection of susceptible individuals, suggests that changing environmental temperature in association with this organism following its excretion deserves further study.

Viruses provide a source of concern in that their fate in sewage effluents and survival in water ultimately used for drinking is not defined. It appears that some of them may escape destruction in both sewage and water treatment operations. Further, dosage factors are not well defined. The agent of infectious hepatitis has not even been identified.

The morbidity of a number of human fecal-borne diseases in the United States is high enough to suggest that considerable environmental fecal contamination occurs. The contribution of individual modes of transmission to this morbidity is essentially unknown. The influence of insanitary foci on the overall incidence of these diseases is also obscure.

Laboratory and epidemiologic methods need further development to help in answering the questions arising from these and similar gaps in our information. Furthermore, the relative importance of socioeconomic conditions and cultural practices in contributing to dissemination of fecal-borne disease of human origin is not fully defined.

CONCLUSIONS (HUMAN FECAL-BORNE DISEASES IN GENERAL)

The worldwide misery and death due to diseases of human fecal origin have been made evident. In the United States, infant mortality and adult morbidity are high in certain populations lacking sanitary fecal waste disposal. These groups are a continuing risk to themselves and to the remainder of the population.

Certain municipal areas, geographic regions and industrial and agricultural activities in the United States have entirely inadequate facilities for the proper disposal of human feces and thereby provide a grave risk to the persons directly exposed. They also constitute chronic reservoirs for the spread of infection beyond their borders. The *Statistical Abstracts of the United States for 1962* (Bureau of the Census, Department of Commerce) states that, of 59,326,000 housing units identified in the 1960 census, 18.2 percent (10,615,332) lacked or had dilapidated plumbing facilities. ["Plumbing facilities" are defined as (1) hot and cold running water inside the dwelling; (2) flush toilet and bathtub or shower inside.] A further 7.8 percent showed deterioration of facilities. Nearly 6 million (10.3%) had toilet facilities other than flush toilets or none at all (see p. XXXVIII); 7.1 percent lacked inside piped water (p. XXXVI).

While other factors — water pollution, lack of washing facilities, human carriers, personal hygiene, flies and possibly other vectors — contribute to the dissemination of these diseases, failure to dispose of human excrement in a sanitary manner and to destroy the contained pathogens is by far the most important factor.

RECOMMENDATIONS

In general, fecal wastes must be disposed of in such a way that they cannot contaminate the immediate environment; that vectors are denied access to them; and that they cannot contaminate food or water supplies. Corrective measures must be applied to the extent that these criteria are not met.

The literature makes clear that sanitary waste disposal alone will not realize optimum prevention of disease. However, little can be accomplished without it. Therefore, the first recommendation is:

1) *Installation of Sanitary Feces Disposal and Treatment in:*

Substandard permanent residential areas in or bordering municipalities

Rural residences

Migratory and construction labor camps and other transient residential areas

Municipalities where sewer overload and treatment plant bypass by sewage is possible

Small communities lacking adequate sewerage or treatment plants

Industrial plants failing to supply or use adequate facilities

Special problems attendant on local economy, geology, water supply, governmental jurisdiction and effectiveness of known methods of disposal and the like will have to be overcome. An existing technology is available to provide sanitary waste disposal and treatment, but other factors may interfere with its application, for example, the costs per capita for sewerage in the small community, the isolation of rural residences and the renovation and sewer installation costs in older community areas. New techniques may be required and this suggests the second recommendation:

2) *Research on Disposal of Fecal Wastes*

While waterborne disposal of feces seems to be the most protective of the present methods of disposal, especially of the immediate environment and individuals involved, the prevention of more widely disseminated disease does not appear entirely assured under present treatment methods.

Definitive, integrated studies on the fate of viruses and other fecal pathogens in disposal systems should be instituted. The efficacy for the destruction of pathogens of various treatment methods, both of sewage and of drinking water supplies, should be investigated.

The use of fecal colon organisms is no longer dependable as the sole criterion for water quality. A bellwether human virus, or bacteriophage, should be identified, if possible, to serve as the *E. coli* of the submicroscopic organisms.

Controlled studies on entire regional waste disposal and water supply systems should be carried out to define fecal-borne disease hazards. They should include integrated microbiologic and epidemiologic studies within the regional systems and among insanitary foci and their environments.

Further, new methods of destruction of pathogens may be required. A recent report by Allen and Soike (*Science*, 1966, 154:155-157, October 7) refers to complete and almost instantaneous destruction of microorganisms, including a virus (the bacteriophage T-2), by electrohydraulic treatment of suspensions of these organisms. The application of such innovations to sterilization of wastes or drinking water should be studied.

Although not applicable at present in the United States composting of night soil is of considerable interest to some countries. Its safety is not fully defined.

New and varied approaches may have to be used to correct deficiencies in fecal waste disposal at places of rural, isolated and transient human occupation. Advance biological treatment systems should be studied for use under these circumstances.

In summary, a large number of studies and research projects are needed to define or refine our knowledge of fecal-borne disease. Presently available techniques of fecal waste disposal and treatment however, are sufficiently dependable significantly to reduce existing morbidity in areas lacking these techniques. Therefore, corrective action does not always require the completion of extensive research projects. Nevertheless, the problems of the future — increasing populations, limiting pollution levels, costs of sewerage, limited space and the like — require that systematic research be instituted in the near future.

3) Public Education

Although some authors feel that much can be done to prevent human fecal-borne disease without education, or that education will accomplish little, especially in the absence of disposal sanitation, these views may be prejudiced by the limits of present educational material or effort.

This education must begin with the very young and in the schools. It is recognized that lack of sanitary disposal facilities and easily accessible water supplies can tend to defeat education, but a demand among the instructed could develop for better sanitation and hygiene. Much bad personal hygiene is due to familial and cultural practices or to ignorance, yet good personal hygiene can exist with rather primitive sanitary facilities.

Joint research efforts are required to develop effective educational material and effective means of teaching and motivating people in guarding against the spread of these diseases.

4) Disaster Waste Disposal

What has been developed so far in this field should be summarized and made a part of the total solid waste body of knowledge. In addition, criteria should be developed and applied within the waste-disposal research program.

Disease Associated with Animal Fecal Waste

Other fecal-borne diseases in which man's domestic animals are the reservoir hosts, or which can serve as hosts, are known to infect man. They comprise all of the major categories of microorganisms noted above under diseases of human fecal origin. Figure 5 shows possible modes of transmission.

GENERAL

Hull[167] lists a number of zoonoses which are transmitted to man by way of animal feces (pp. 915-924). He mentions first, the intestinal protozoan, *Balantidium coli* of the pig (pp. 435-436); second, some of the helminths such as the pig Ascaris and animal tapeworms (p. 444); third, the hydatid worm, *Echinococcus granulosus* (p. 445); fifth, the salmonellae for which animals are the main reservoir (p. 210) etc., and fifth, a small number of other infective agents. He states that although there are large numbers of species of protozoa found in vertebrate hosts, very few are known to infect man. The species of helminths of animals transmissible to man were reviewed in 1962 by Reed and McMillan.[558]

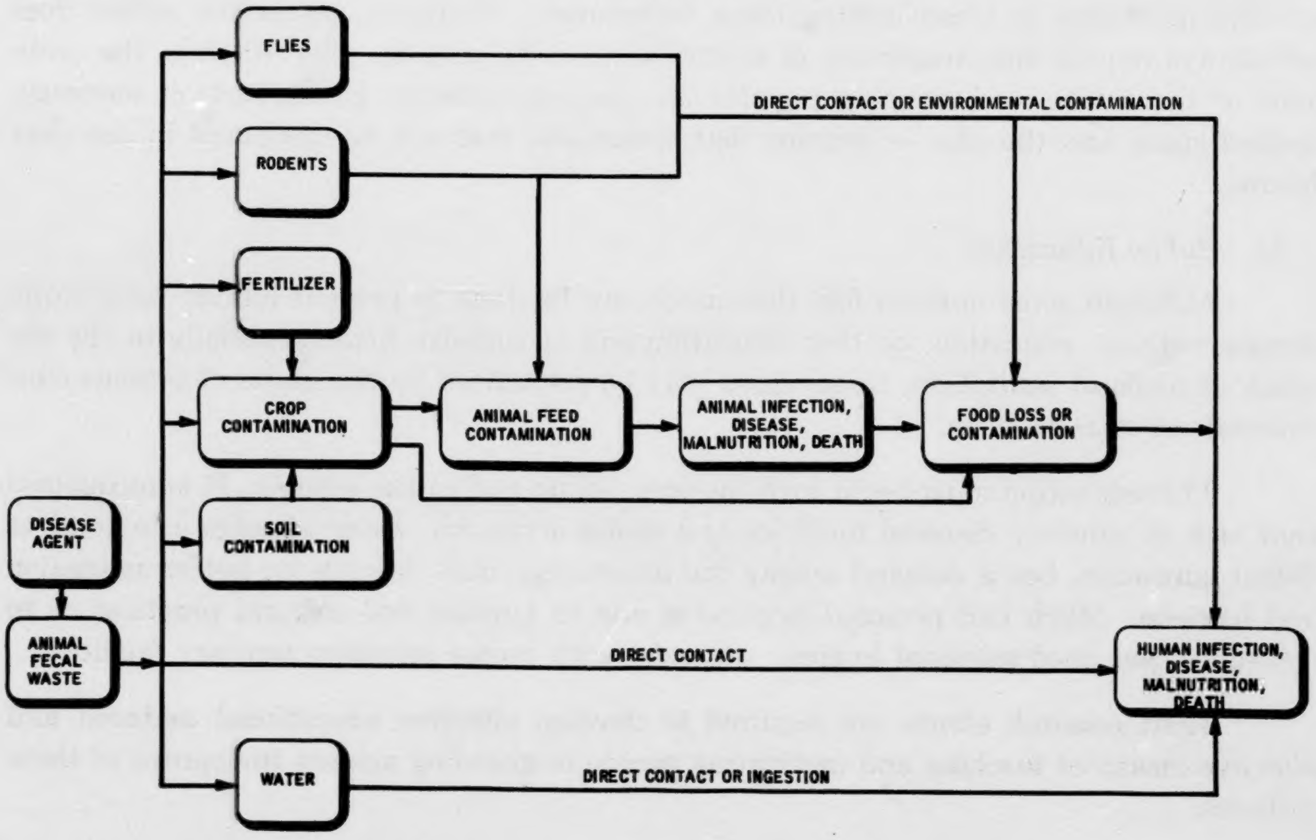
FLIES
RODENTS
FERTILIZER
DIRECT CONTACT OR ENVIRONMENTAL CONTAMINATION
CROP CONTAMINATION
ANIMAL FEED CONTAMINATION
ANIMAL INFECTION, DISEASE, MALNUTRITION, DEATH
FOOD LOSS OR CONTAMINATION
DISEASE AGENT
SOIL CONTAMINATION
ANIMAL FECAL WASTE
DIRECT CONTACT
HUMAN INFECTION, DISEASE, MALNUTRITION, DEATH
WATER
DIRECT CONTACT OR INGESTION

FIGURE 5. Animal fecal waste/disease relationships (postulated).

In view of the breadth of subject, it will be impossible in this report to review all of the diseases in this group individually; it must suffice to show the complexity of the problem of zoonoses in its association to waste by reviewing the subject of salmonellosis and referring only briefly to some of the others. In this section, the salmonelloses of human origin will not be discussed.

The relationship between flies, wastes, and the salmonelloses are covered elsewhere in the report. In summary, flies are known to transport the bacteria; in some cases, the bacteria have been shown to multiply within the fly.

EVIDENCE

1) Salmonellosis. In 1947 the medical research council of London reported on the importation of strains of salmonellae to Britain during World War II.[547] These strains were noted not to have been found in Britain previous to the war. They were traced to imported dried egg products from the United States, Canada, and the Argentine.

The report concluded that "the epidemiological and bacteriological evidence submitted in this report leaves little doubt that the introduction into this country of American spray-dried egg led to a considerable increase in the amount of food poisoning and other forms of *Salmonellae* infection in the human population, and to infection of some, at least, of our farm animals". In the United States, of 53 outbreaks of poisoning occurring in 1951 in which "poultry, meat, and eggs, were found or suspected to be the vehicle of infection," eight proved to be caused by salmonellae.[53] In that year, a total of 850 clinical cases were reported.

In 1953, in a study of a California county, Watt[539] and others found that salmonellosis was a relatively infrequent infection in all the California groups studied. The prevalence rate found was only 0.4 percent. The study groups were in labor camps and in town fringe areas.

Hull,[167] reporting on a study by McCullough and Eisely, stated that twelve strains composing six types of salmonellae all caused illness in human volunteers when sufficient numbers of the bacilli were fed. Within these types and strains, however, the infective dosages ranged very broadly. Hull stated that most domestic animals and commensal rodents harbored the infective agents. Man was also a host and could spread the disease through his own feces. Hull also discussed modes of transmission through contaminated meats and eggs. He stated that Hobbs in England incriminated boneless meats, carcass meats, meat and bone meals for fertilizers, and animal feeding stuffs as providing sources for salmonellae contamination. Canned meat, packaged mixes for the kitchen, bakery goods, dried eggs, and many other food substances have also been determined to be vehicles for transmission of the disease agent (pp. 210-231).[167]

Jellison reported in 1951 on attempts by others to transmit salmonellosis by means of fleas.[156] The pathogens were noted to survive in the fleas for up to 96 hours, but transmission by bite was not demonstrated.

In 1958, Schliessmann and others[545] were able to demonstrate only rare salmonellae isolations from over 11,000 rectal swab cultures collected from mining camp populations in the United States.

In 1958, one text[682] stated: "The source of all *Salmonellae* infections is a reservoir or organisms living in the tissues of human beings or animals. Infections occur through food, milk, or water contaminated with infected feces or urine, or by the actual ingestion of the infected animal tissues. . . . Infection with most *Salmonellae* apparently requires the ingestion of large number of organisms, but in the case of *S. typhosa* relatively few bacilli are sufficient to cause typhoid fever." The means by which water may be contaminated by infected feces is discussed, and human infection due to shellfish contaminated by polluted waters is noted.

In 1959, Smith discussed the transmission of salmonellosis in association with slaughterhouse procedures and the part played in salmonellae infection in animals by infected feeding stuffs.

Harvey and Phillips[695] noted in 1961 that a large bakery was demonstrated to be regularly contaminated with salmonellae. Many serotypes were found in abattoirs. They stated: "Parallel examination of abattoirs, swabs, and excreta from human infections for *S. typhimurium* . . . provided information of the geographically local nature of much sporadic illness due to this serotype . . . The phage-types of *S. typhimurium* isolated from the slaughterhouses not infrequently corresponded with the strains isolated from human infections."

In 1961, Anderson and others[342] reported on a two-month period in 1958 in which 55 separate food-poisoning incidents, comprising 90 cases due to *Salmonella typhimurium*, phage-type 20a, were reported in Southeast England. There was one death. It was stated that "though not conclusive, the evidence strongly suggested calf meat as the vehicle of infection in at least three-fifths of the incidents."

In 1961, Dauer[168] stated that in disease caused by salmonellae organisms, "The wide distribution of the many types of this organism in many species of animals with which man has contact or may use as food makes it difficult to prevent transmission to man . . . In addition to the animal sources of infection, human carriers and patients have many opportunities to contaminate food . . . it can be argued persuasively that there is a real need to improve the reporting of both waterborne and foodborne illness as the first step of the development of effective control measures. There is also real need to study more intensively and extensively the epidemiology of . . . *Salmonella* infections because these causative agents are widely distributed and their ecologic factors are complex."

In 1962, Ayers[316] reviewed the material on salmonellosis due to contaminated food. He noted the worldwide distribution of salmonellae types by the importation of animal and human food stuffs.

Pratt and Wiseman stated in 1962 in regard to salmonellosis: "Meat and other foods contaminated by unsanitary conditions, including infective feces of rats and mice, are generally considered to be the prime source of human infections. It is possible that human infection can be contracted directly from the bite of an infected flea or from food stuffs contaminated with their feces."[398]

In 1962, Galbraith and others[104] stated that "survey of garden fertilizers in England revealed 13 percent contamination with salmonellae." Concern was expressed for permitting such fertilizers to be sold in shops selling food for human or animal consumption. It was stated, however, that "the risk [of spreading infection] is probably small."

The hazard of using fish wastes and offal was discussed in 1962 by March and others.[583] In this report, Shewan stated: "From the small amount of data available, it is clear that fish and fishery products can be the vehicle for all of the more important types of bacterial food poisoning (including salmonellosis)." Shewan stated further that "all the available evidence shows . . . fish normally do not suffer from salmonellae . . . infections. . . . They may well carry these infections if caught in polluted waters but this risk is of much less importance in marine fish than in fresh water species for the dangers of pollution in rivers and lakes can be very real." This author summarized data on infection of fish taken from rivers of probable high contamination with the disease organism, and added that "although it is not suggested that the fish suffer from these infections, they do appear to act as intermediate hosts for man, with the lakes themselves acting as immense reservoirs." He stated also that fish caught in the open sea are free from salmonellae and the shigellas. "It is to be expected, however, that once the fish have been handled on ship and on shore, some contamination from human sources is likely to ensue. Fecal contamination, as evidenced by the presence of *E. coli* and fecal streptococci, increases during handling and processing." Shewan also said that "in recent years there has been an outburst of salmonellae epidemics, chiefly in West Germany, which could be traced to imported fish meal. These salmonellae spread to humans via livestock."

In this same reference,[583] Kawabata noted the high incidence of salmonellae food poisoning in Japan where fish consumption is high. Cultural habits, particularly those of eating fish raw, are noted as playing an important part in these infections. Guelin noted contradictory findings among authors reporting prior to 1962 but stated that, "as a whole, the results show the accidental character of the presence of Enterobacteriaceae in fish." He then reported on experiments on the artificial contamination of a single species of fish with *E. coli.* He found that the contamination by both the bacteria and by coli phages was not of long duration, but stated that it would be necessary to repeat the experiments with other species of fish and other enteric bacteria. In the same reference, Buttiaux stated: "Recent publications have proved the negative results of studies undertaken to find salmonellae and other enterobacteria of fecal contamination in marine fish caught in the open sea. Certain observations indicate, however, their presence in fish when they are marketed fresh or dispatched from the filleting or icing establishments." He said that "edible oysters, mussels, and other shellfish are, on the other hand, very often infected by salmonellae, because the indispensable sanitary precautions are neglected in the cultivation areas." In regard to the survival of salmonellae and other enterobacteria in seawater, he stated: "The data hitherto collected are still highly inadequate. . . . It is generally considered that salmonellae are no longer found where *E. coli* and coliforms are controlled or have disappeared. This reasoning results from the neglect of two basic principles during experimental studies." He stated also that "[Some] publications show that it is not exceptional to find viable salmonellae in seawater contaminated by polluted water . . . Other authors have confirmed that fish never contain coliforms when caught in the open sea . . . stated that on the other hand everybody recognizes that fish may become infected through their intake of food when staying in seawater contaminated by sewage water or polluted rivers. This rule applied also to freshwater fish." He noted the problem of poor sanitation in the spread of salmonellae to fish, and added that "salmonellae carried into the sea by an outflow of untreated sewage effluents may easily contaminate mollusks and fish in adjacent waters. . . . Those [fish] caught in polluted littoral zones may be contaminated by bacteria. Most salmonellae in fish are procured in the handling, transporting, or processing operations when these are carried out under inadequate sanitary conditions. Edible mollusks concentrate salmonellae in their bodies . . .

such mollusks are readily infected when sewage effluents are not strictly controlled in coastal areas where they grew."

Salmonellae infection and prevalence are noted to vary in various regions.[694, 550, 537]

In 1963, McCoy[103] stated: "Of the pathogenic organisms present in crude sewage, which consists essentially of human excreta suspended in the waste waters of the community, organisms of the Salmonella group, which are widely distributed in man and animals, are by far the most common. . . . salmonellae of animal origin in towns are derived from trades and industries processing animals and their products for human or animal use. These sources include: abattoirs, butcher shops, the make-up meat industry, poultry processing plants, egg breaking plants, bakeries, tanneries, knackers, knacker's premises, animal fat extraction plants, meat-meal and bone-meal plants, animal feeding stuffs plants, and fertilizer plants." This author stated that salmonellae can survive on vegetation and in the soil from three to seven days and up to 280 days respectively. He said "under natural conditions the disappearance of salmonellae from polluted vegetables is slow and inconstant . . . experimentally, the numbers of bacteria contaminating the vegetation or soil seemed to be the most important single factor in the disappearance of salmonellae from them."[103]

The proceedings of the March 1964 National Conference on Salmonellosis[693] discusses the pervasive worldwide problem of the salmonellosis. In this reference, Canadian authors Yurack and Best of Canada state ". . . since 1955, there has been a steady and marked increase in the reported incidence of human Salmonella infections other than typhoid and paratyphoid . . .". They concluded that human salmonellosis in Canada was steadily increasing and that greater numbers of salmonellae were being isolated from animals in a wide variety of processed human foods.[693]

Newell[693] noted that salmonellosis was an important disease in the United States and in most countries of the world but that "the real human mortality related to this cause is unknown." He stated that "most salmonellae can infect most hosts, but the dose of organisms required to cause an infection in the occasional host may be very large indeed." In one cycle of animal-to-man infection, he noted that an intermediate stage was necessary, allowing multiplication of the organisms to take place. He thought it was possible for some human infections to be caused by air-borne transport of the organism. He concluded that "the prevention of man-to-man transmission by environmental methods or by the control of excreters or carriers is unlikely to result in a large decrease in human salmonellosis. The education of whole populations about the dangers of certain uncooked foods kept at room temperatures is unlikely to be successful in the short term without a major change in food preparation and eating habits, and is impractical. That certain direct and indirect contacts from animals to man can be made Salmonella-free has been demonstrated in the United Kingdom egg legislation of 1963. However, it is improbable that all such contacts can be policed in this way. Therefore, the most workable and immediate solution must be the selective breaking of the animal cycle starting with the most controllable domestic animals whose specific salmonellae are most important to the human population of a particular geographic area. I consider that such a selective attack upon the animal cycle is both possible and economical in this country. The host of first choice would probably be domestic poultry" (p. 42). In the same work, Thatcher (p. 61) notes the problem of the modern mass-producing convenience food industry in the spread of salmonellosis.

Steele and Quist (p. 72) state that "there is a high prevalence of Salmonella infection in a variety of pet animals including dogs, cats, birds, and reptiles. Little is known about the potential spread of salmonellosis from animals to stockmen or pet owners by contact."[693] These authors also state (p. 73) that "to significantly reduce the human incidence of salmonellosis, it is imperative that attempts be made to control the infection in fowls and other animals." Pomeroy and others, noting the spread of Salmonella through contaminated feeds, state: "Because of the wide host range of Salmonella and ubiquitous nature of the organism, consideration should be given to the development of sanitary methods that will assure the final complete feed to be free from pathogens. This may require the 'pasteurization' of the feed in the final stages of manufacture."

In closing this particular conference, Langmuir noted the large number of ecologic and epidemiologic questions regarding this disease that remained unanswered. He concluded that better case reporting, tighter surveillance, and detection and management of carriers were needed, as well as the elimination of Salmonella from foods and feeds, and more cooperation in international surveillance.

In 1964 various authors[324] discussed and published the general topic *The World Problem of Salmonellosis.* They referred to an ever increasing spread of different species among both animals and humans. Fey quotes Newell (p. 172) as demonstrating convincingly that the source of Salmonella (always apart from man-adapted typhoid–paratyphoid) ultimately lies in animals and that the long-range prevention of human salmonellosis is to be sought in an interruption of animal-to-animal contact and not of man-to-man. The practice of man in herding and feeding animals as an agent in causing the spread of the disease is noted (p. 178). The importation of new strains of the bacterium, particularly from tropical countries, is also noted (p. 266). The contrived pathways of infection leading from animal to man, and resulting in outbreaks of disease, is referred to in connection with this disease (pp. 271-272). The importance of rodents in transmitting the infection among domestic animals is referred to (p. 273).

In this same reference, it is stated that rats are recognized as being generally fairly susceptible to Salmonella infections and develop severe typhoidal disease. They live in very close association with human life. There are, therefore, many possibilities, not only for human, but for domestic animal infections derived from rats carrying Salmonella and transferred by direct or indirect contacts. In particular, the behavior of rats is an important factor in the contamination of food utensils with infected excreta, etc.

The British, reporting in 1964 on a study of various food processing factories and shops, made the following comment: "It was often shown that the same serotypes or phage-types were occurring in abattoirs and in human cases in an area at the same time."[133] In many instances of food poisoning, meat or meat products were suspected as being the vehicle of infection. Cattle were implicated as the most important conveyors of salmonellae, whereas sheep were not considered to be a source. In 1964, Rosenstein[571] reported on Salmonella infections among infants and children in a children's home, and Hendrickson[546] reported an outbreak of gastroenteritis from *Salmonellae heidelberg* which was found to be contained in packaged angel-food cake mix used in the kitchen of the construction camp where the outbreak occurred. Hendrickson also reported on an outbreak of hospital infection with the same organism, apparently introduced by a surgical patient. Schneierson and Bottone[549] in 1965 reported that salmonellosis infections were only about one-fourth as great as Shigella infections among hospitalized patients in an underprivileged community. VanDerSchaaf and Atteveld in 1965 reported on the almost universal contamination of water in the Netherlands by salmonellae organisms, in spite of the widespread biological treatment of sewage in that country.[80]

2) Balantidiasis. Craig and Faust[319] in 1940 stated: "Man is infected by swallowing the cysts of *Balantidium coli* in food contaminated by fecal material or by direct transference of infected pig's feces to the mouth through soiled hands in handling pigs or in slaughtering operations. Considerably over 25 percent of the recorded cases of infection give a history of direct contact with pigs, while the use of the excrement of pigs as fertilizers may be the source of some infections" (pp. 217-218).

In 1942, Strong[198] stated that this disease was a much rarer infection than amebic dysentery in Texas, and in North and South Carolina. He stated that cysts were transferred to humans through the handling of infected gastrointestinal tracts of pigs, the eating of raw sausage, or from pig manure (p. 445). He noted that the cysts survive for weeks in moist feces (p. 452).

Hull's book[167] states: "Human infection is most probably acquired from the reservoir host as a result of contamination of food or drink with pig droppings which contain the encysted organisms. Patients with balantidiasis often give a history of close association with pigs. . . . Once the infection has been established in man it may be either chronic or acute and at times develops in epidemic form. Furthermore, there is no satisfactory drug for the eradication in the human host."

In 1962, Hoare[555] stated that human infections from this disease were relatively rare, citing that "only several hundred cases have hitherto been recorded throughout the world." He noted that the ciliate was a common parasite of domestic pigs, over 90 percent of which may be infected in some countries. He stated that the disease "is practically an occupational disease among pig farmers, swine herds, slaughterers and sausage-makers." He added that there was some evidence that this disease could also be transmitted from man to man.

3) Diseases Associated with Dog Feces. In dealing with the general subject of diseases arising from animal fecal wastes, one would be remiss in not discussing those of the dog. In view of the very close relationship of dogs with humans, and especially with children, the present urban practices in regard to disposal of dog feces deserve serious consideration.[319]

The dog suffers a number of diseases transmissible to man through the agency of its feces.[167, pp. 893-894, 915-924] The following list is taken from Hull:[167]

Amebiasis; ancylostomiasis (dog hookworm); balantidiasis; echinococcosis (hydatid cyst); fasciolopsiasis; larva migrans visceral; larva migrans, cutaneous; opistorchiasis; paragonimiesis (lung fluke); salmonellosis; schistosomiasis; strongylodiasis; trichinosis.

Some of these [e.g., a nematode disease, dog hookworm,[167, p. 896] and the treniatodes] are apparently of little importance as human infections in the United States.[167, p. 896]

It is not possible to estimate the incidence of human disease due to dogs through this route of infection.

DISCUSSION

In developed countries, the relative incidence, prevalence, and severity of human infection due to animal fecal wastes is low if one looks only at reported outbreaks. The suspicion is that the amount of disease is actually much higher.

If infection is to be reduced, consensus singles out animal-to-animal spread as the primary point of attach — especially among food-source species. Human practices in animal-source food production or processing, food storage and preparation, and food-waste disposal are major factors in the chain of infection. Contamination of meat in the slaughter of animals and fowl or dressing of fish, of egg products for packaged foods, and of other foods which support the growth of salmonellae typifies the extent of the prophylactic problem. Yet animal wastes must in some way be controlled; apparently success in preventing human disease by attacking the human aspect alone is less likely, according to some authorities.

CONCLUSIONS

The fact itself, and some of the modes of transmission to humans of diseases associated with animal fecal wastes, have been established. A few of these modes suggest that waste control may be preventive of a limited amount of disease incidence in humans in the United States.

One can refer to 'animal sanitation' in only a very relative way. As a goal, it must await large-scale mechanization of animal breeding, controlled feeding and housing of stock, and sanitary methods of feeding inclusive of sterilization of animal feed. While all of these techniques are seen in practice at present, they are seldom found together. Only inclusive application of these or similar practices, however, can help to prevent disease through animal waste control.

RECOMMENDATIONS

The vast amount of animal feces produced in this country demands further study of a means for its management. Sanitary aspects of disposal should be included in such a study. Comparison of existing composting methods should be extended to determine their capacity in operational, as opposed to laboratory, status so as to destroy pathogens and prevent vector propagation.

Public education should be directed at proper disposal of pets' feces. The hazard of well contamination by animal feces in relation to infant methemoglobinemia needs publicity in rural areas. The biological hazards of animal wastes require that additional education of farmers and food processors be instituted; stricter surveillance of food handling and processing is an obvious requirement.

Rodent-Borne Disease

GENERAL

The following is a list of zoonoses associated with the rat (taken from Hull, [167, p. 915]: echinostomiasis; hemorrhagic septicemica; histoplasmosis; lymphocytic choriomeningitis; plague; rat-bite fever; rat-mite dermatitis; rat tapeworm infection; Rocky Mountain spotted fever; salivary gland virus infection; salmonellosis; schistosomiasis; bilharziasis; sporotrichosis; swine erysipelas; trichinosis.

To this list must be added leptospirosis, leishmaniasis, relapsing fever, tularemia, rickettsial pox, murine typhus, and perhaps other diseases.[166, 398] The house mouse is a host of rickettsia.[43, 520, 574]

Commensal or domestic rodent species, as these adjectives imply, live in close proximity to man and his domestic animals, thus satisfying a primary requirement for transmission of disease to man.[330, pp. 279-80, p. 370; 558, p. 356]

In addition, the habits of commensal rodents are such as to permit contamination with their excreta of man's food, clothing, utensils, or to subject man to direct contact or attack.[324, p. 11, abstract]

To associate rat-borne (domestic rodent) disease in man with solid waste, it is first necessary to associate the rodents with the waste. It must further be demonstrated that commensal rodents transmit disease to man.

Finally, all crucial epidemiological links in the postulated rat-to-man chain of disease must be demonstrated.

POSTULATION

The commensal rat is a known source of zoonoses (diseases of animals transmissible to man), and thrives wherever carelessness in food waste handling and disposal is found. Because of its habits and close association with man, it exposes man to various disease agents which are transferred by direct contact, ectoparasites of the rat, or by contamination of the human environment.

Figure 6 illustrates postulated pathways for human infection by the disease agents of plague for which the rat is a host or carrier, and can serve in part to demonstrate pathways of some other rat zoonoses.

EVIDENCE

Rats are attracted to, and multiply in, refuse and associated residues.[49, p. 21; 68, 91, 150, 165, 181, 465, 285, 291, 334, 379, 458, 565, 574, 544, p. 182]

Rats have also been found at waste disposal sites such as stabilization ponds[68] and poorly operated sanitary landfills.[91]

As recently as 1964, citizens of the United States were observed throwing their garbage and refuse along the roadside as they drove from rural homes to their work.[150] Many smaller communities in the United States still dispose of their solid wastes in open dumps.[91, 231]

Rats harbor ectoparasites which are known vectors of disease, and exchange parasites with other animals that are hosts to disease agents transmissible to man[165, 313, 566] Fleas, ticks, and mites frequent rodent nests and burrows of both domestic and wild rodents and are the means of transferring disease from one rodent to another.[162, 320] These anthropods are frequent feeders on man when in proximity to him. Such proximity occurs when man invades the wild reservoir territory or when domestic or wild rodents invade man's domicile.[165] The latter situation is encouraged by careless waste disposal, although improper food storage or feed handling practices can attract rodents (p. 2).[520]

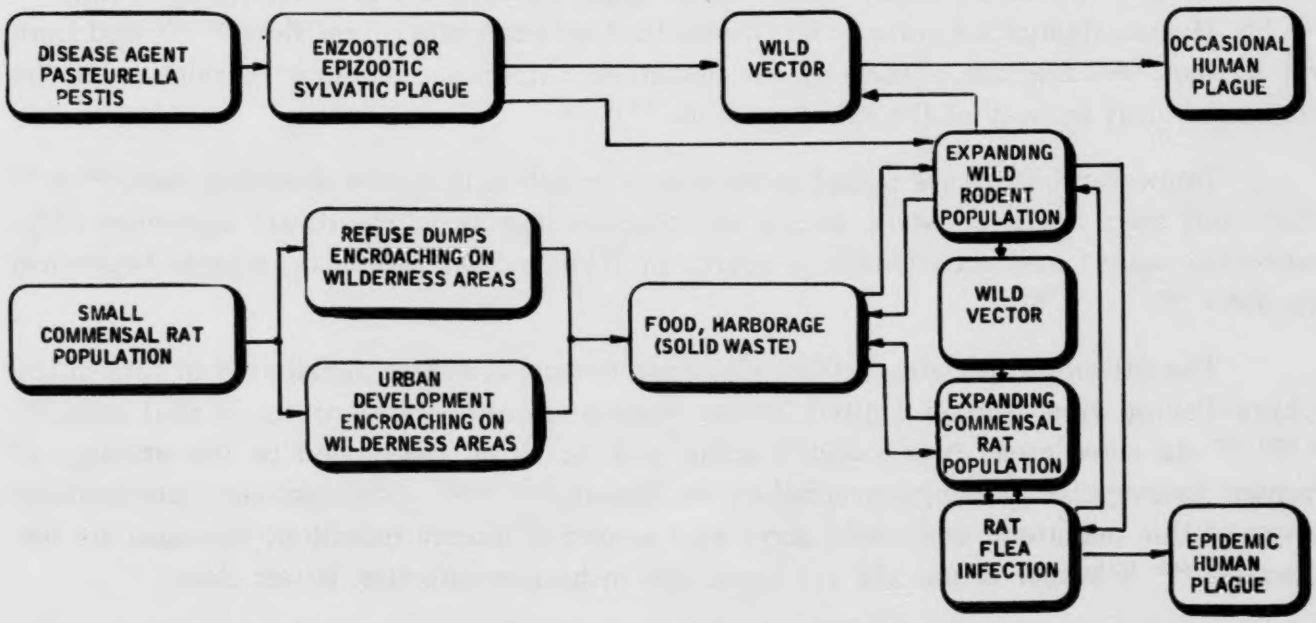
DISEASE AGENT PASTEURELLA PESTIS
ENZOOTIC OR EPIZOOTIC SYLVATIC PLAGUE
WILD VECTOR
OCCASIONAL HUMAN PLAGUE
EXPANDING WILD RODENT POPULATION
REFUSE DUMPS ENCROACHING ON WILDERNESS AREAS
SMALL COMMENSAL RAT POPULATION
FOOD, HARBORAGE (SOLID WASTE)
WILD VECTOR
URBAN DEVELOPMENT ENCROACHING ON WILDERNESS AREAS
EXPANDING COMMENSAL RAT POPULATION
RAT FLEA INFECTION
EPIDEMIC HUMAN PLAGUE

FIGURE 6. Solid waste/plague pathways (postulated).

Rats can serve as a major threat in transfer of zoonoses to man. While they are not, at present, reservoirs of plague in the United States, they can serve potentially to spread sylvatic plague which is now endemic throughout this country.[165, 569] The mechanism is discussed under "Plague" and, in general, is similar for other arthropod vectored disease of rats and wild rodents.

Rats can transmit disease by direct contact, as for example, in causing rat-bite fever in man.[53, p. 198] In addition, by soiling food, clothing and eating or cooking utensils, rats can probably cause human salmonellosis.[544, 324, p. 273, pp. 10, 11 (abstract); 168, 398] They are known to be involved in the Salmonella infection cycle in domestic animals.[324, p. 273]

Rat fleas can be significant pests and can cause flea or mite dermatitis.[319, 112, 156] Rat fleas are also true hosts of several tapeworms of lower animals which are known to be transmitted to man, especially children.[398, 156]

At times, rats have been thought to be an agent in the transmission cycle of human trichinosis. As recently as 1956, a significant number of trichina-infested rats were found in village and city dumps in the American Midwest. The investigators state that the role of rats and wildlife in trichinosis of swine is difficult to determine.[508] They postulate, as possibilities, fecal transmission or direct transmission where pigs eat infected carcasses. They were not successful in fecal transmission efforts, but quote Spindeler as having obtained success in such transfer. Other authors state that there is no evidence that the rat plays any part in the pig cycle, and by implication, none in the human cycle[558, p. 369] — at least in the United States.

Human leptospirosis has been traced to rats.[526, 517, pp. 901-908] Leishmaniasis, a disease rare in the Western Hemisphere and not found in the United States, has been found in domestic rats of two genera,[556, p. 316] and has been experimentally transmitted to rats.[319] Rickettsialpox in man has been traced to the house mouse; it is transmitted by a mite.[43, 399, 574] Murine typhus is known to be transmitted between rats by rat fleas[177, 156] and from rat to man.[156] Infection of man by the nematode *Capilloria hepatica* (a relative of the trichina worm) by way of the rat is possible.[558, p. 375]

Domestic rodents are stated to be donors in helminth cycles involving man.[558, p. 356] Rats and mice are a constant source of infection in man of the dwarf tapeworm *(Hymenolepis nana)* and occasionally a source of *Hymenolepis diminuta*, a large tapeworm (p. 366).[558]

The rat lungworm, *Angiostrongylus cantonensis*, is widely distributed in rats in the China–Pacific area, and in United States possessions and protectorates in that area.[346, p. 558; 377] In some areas it is found in man, and has been implicated in the etiology of human eosinophilic meningoencephalitis in Hawaii.[558, p. 378] Mollusks are intermediate hosts of this nematode and could serve as a source of human infection; pigs also are implicated.[346] Whether or not the rat has a role in human infection is not clear.

Tularemia is said to be naturally present in wild rodents, including wild rats[379, [338, 156, 198]; the natural vector is the woodtick, *Dermacentor andersoni.*[198] Rats are stated to be only slightly susceptible, however, to the disease.[198, p. 716] Experimentally, fleas and mites have been found to be infected[398, 338] and to transmit the disease agent,[198, pp. 714-715] However, they have not been shown to be important in the transmission of tularemia.[156, 398] The tropical rat mite was shown to harbor the organism of tularemia and, in some instances, to transmit it to the next generation of mites; it also transmitted tularemia to normal mice on being crushed orally by the mice, but not on biting the mice.[338] The incidence of human tularemia recently has been described as paralleling the population of "mouse-type rodents" during and after World War II in Russia; the author considers bloodsucking arthopods worthy of investigation as vectors.[379] In Russia, it is claimed that the basic intermammalian mode of transmission is via the tick.[380]

The rat is definitely associated with murine typhus; its ectoparasites — mite, rat louse and rat fleas — spread the agent from rat to rat.[517, p. 80; 156, 177, 319, 394, 398] The tropical rat flea, *Xenopsylla cheopis,* is the most important vector for rat-to-man transmission.[339; 517, p. 80] Fleas may not be the only, or even the main, transmitter, and rat urine has been implicated via dust in human infection.[156, p. 400]

Only 42 cases of human murine typhus were reported in the United States in 1961[520] and 29 in 1964.[351] This represents quite a drop from the 5,193 reported cases in 1945.[517, p. 80] The dramatic reduction in human murine typhus is believed to be due in all probability to the coincident rodent control measures.[398]

In South Africa, the disease has apparently been introduced in recent years, and is resulting in occasional human cases.[339] Here, too, a domestic rat, *Rattus rattus,* is the reservoir. The disease is considered more an occupational than a domiciliary disease.[319]

Lymphocytic choriomeningitis is a viral disease in man. The reservoir is the mouse. Hypothetically, the virus is conveyed to man by way of contaminated food or dust; human cases have been associated with infected mice.[167, p. 726]

Hull[167, p. 922] lists rats as a factor in the transmission of Rocky Mountain Spotted Fever (RMSF) to man, but does not support the association in the text. A number of ticks are both reservoirs and vectors; others can be infected experimentally.[167, p. 701; 335] The vectors appear to prefer large animals, both wild and domestic. In Maryland, dogs and field mice are said to be important in the epidemiology of the disease in that state.[341] Experimentally, the rat has been found susceptible to the infeciton.[167, p. 703] However, no evidence was found for implicating the rat in human infection. In the period 1960-64, 229 cases of this disease were reported in the United States.[351] In none of these cases is the rat mentioned. The postulated mechanism for transmission of RMSF to man by way of the rat can be discussed in the general case of tick-borne disease. In the United States, species of ticks are vectors of RMSF, tularemia, relapsing fever, Colorado tick fever (CTF), and tick paralysis[516, 157, 335] The species for RMSF important to human infection are *Dermacentor andersoni* and *D. variabilis*[167, p. 701] for tularemia. A number of different tick species are vectors[167, p. 592] for relapsing fever in which numerous subspecies of Ornithodoros are involved.[167, pp. 669–673; 198, p. 331] For CTF, *Dermacentor andersoni* is the only important vector[167, p. 789]

The rat is not a normal host for any of these diseases except perhaps relapsing fever in Panama.[167, p. 671] The ticks themselves are commonly the main reservoir of the disease agents.

It is possible that infected ticks from any of the wild reservoirs could feed on domestic rats and transmit the respective pathogen to the rat. Except for perhaps the case of relapsing fever in Panama noted above, there is no evidence that this occurs in nature, or, if it does, that the rat could then be a source of infection for man. The vector of RMSF in the Eastern United States — the dog tick — could infect the domestic rat, but again there was found no evidence of such transfer.

Aside from the domestic rat, however, the small wild rodents attracted to wilderness cabins, camps, or lodges by carelessly disposed garbage could convey infected ticks to man's vicinity and thus bring about human infections. Deer mice and squirrels in Montana for example, have been shown to have been infected with CTF serologically and culturally.[157, 174] Agents resembling those of relapsing fever were found in chipmunks and Tamarack squirrels in California.[161] The rodent and human strains were found to be identical, and the rodent strains are directly transmissible to man. It can only be assumed that human cases arising from the endemic foci associated with these rodents were due to exposure to rodents.

Although echinostomiasis and histoplasmosis are tabulated by Hull[167, p. 918] as zoonoses associated with the rat, there is no case to be made for rat-to-man transmission. Transmission to humans of the remaining zoonoses listed as rat-associated by Hull was not demonstrated in this study.

DISCUSSION

The commensal rat and his feral cousins have been shown to carry and transmit disease agents infectious for man. They breed in, or are attracted to, food wastes. The commensal rat can propagate in large numbers where food wastes and harborage are available. (There appears to be some difference of opinion as to how large the waste-dump rat populations can be — see references 181 and 622.)

The interchangeability of rodent ectoparasites under certain circumstances can bring sylvatic disease conveyed by these vectors much closer to man through the agency of the rat.

Furthermore, the rat by its habits can contribute to the contamination of the environment of man and his domestic animals and thus to a double threat to man by way of infection of his animals.

Considering the adaptability of the rat, the numerous sources of food other than wastes, and the obscurity of its contribution to human disease incidence, the relative importance of solid waste toward this contribution is equally obscure. However, the fact that there is a positive association, and that the rat is a dangerous enemy to man, have been made evident.

CONCLUSIONS AND RECOMMENDATIONS

There is continuing threat of human infection by rat borne zoonoses in the United States; while presently of relatively low or unknown degree, its containment requires a higher level of waste management and rodent control than now exists, if only to decrease risks of epidemics in disaster areas.

While the control of wastes is of greatest importance in controlling rats in centers of large human populations, wastes are not the only source of food for these rodents. Carelessness in warehousing of food supplies, in storage of food in the home, and in storage and distribution of animal feeds, and the ability of the rat to find forage in the wild, complicate the control problem.[165, 465, 565] In fact, some investigators feel that these sources are more important to the propagation of rats than waste. It will be necessary, therefore, to coordinate waste and other rodent-control studies and measures.

The prolific breeding capacity of the rat, however, permits its spread from garbage dumps to homes, farms, and other areas where it may find other food sources, and presents a higher risk of conveying infection by reason of its nearness to human habitats.

Various authors have advised adoption of the following procedures:

Development of more effective waste handling and management methods

Public education in food storage and garbage disposal

Increased research on domestic rodent eradication

Increased research on arthropod vector eradication

Development of more effective means of immunization against rodent borne diseases

The wide distribution in nature of many of the zoonoses associated with the rat will not, of course, permit elimination of the basic source of these diseases even if the rat is controlled. However, the control or eradication of rats will drastically limit the opportunities for human infection from the disease agents represented.

SPECIFIC DISEASE

1) Plague. General. Because plague has been so devastating in the history of man, and because the rat and its parasite, the flea, are suspect in other diseases transmissible to humans, plague is chosen as the first of two specific rat zoonoses to be considered in association with solid waste.

Postulation. Plague is capable of transference to human populations by the sequential events diagrammed in Figure 6 in which solid waste is a major factor.

Evidence. The commensal rat is firmly established as a source of human bubonic plague.[172, 167, pp. 527-532, pp. 902-903; 517, p. 197; 190, 313, 319, 573] It is found throughout the United States and the world[682, p. 417] In 1947, a rat cenus showed a ratio of 1 rat for every 36 persons in New York City, and 1 for every 15 people in Baltimore.[167, p. 903] Rats are especially common where food is plentiful, particularly around open dumps[6]; (also see references, section on rodent borne disease). Their usual ectoparasites infest wild rodents, and they in turn can be infested by wild rodent ectoparasites.[162, 164, 313, p. 142; 566, 568, 682, p. 418] Transmission of plague from wild rodents to rats has been demonstrated,[569, 570] and has resulted in human plague via the rat.[172, 164, 570]

The rat is highly susceptible to infection with the plague bacillus and following infection often dies. The vector flea then leaves the body of the rat in search of a new host and, given the commensal status of the domestic rat, this host often is man.[517, p. 201] If a large rat population is present, the "dosage" of bacilli thus made available is high, and human disease much more probable, than if the rat/flea population is low.

The vectors are the interchangeable ectoparasites.[190, 191, 320, 340, 398, 569, 517, p. 199] Many flea species are known to be infected with plague bacilli.[313, p. 142; 398, 567, 570, 573]

Urban or port plague apparently was introduced about 1899 into the West Coast of the United States, and by 1908 was established in western wild rodents.[573, 156, 172, p. 456] However, some authors feel that sylvatic plague has existed among American rodents and infected the American Indian in prehistoric times.[690, p. 1566] Since then, plague was increasingly found among wild rodents through the country[162, 190, 569, 682, p. 418] and the world.[573, 517, p. 199; 167, pp. 534–555, 563–568] Subsequently, it reappeared in rats from time to time.[162, 313] These wild rodents supply what is probably an ineradicable reservoir of plague.[165, p. 43; 164, 162] Plague has also been passed from wild rodents to humans.[152, 319, 567] The flea vectors apparently increase in population with increase in numbers of the rodent hosts.[566, 682, p. 419] They are found on man in association with human plague.[568] (An excellent review of plague in the United States is given in reference 690.)

Discussion. All of the postulated links in the chain of infection involving a solid waste/human disease relationship have been demonstrated. How meaningful are they to an outbreak of epidemic of pandemic plague in man? In the last 30 years, only a handful of human cases of plague has been reported in the United States[166, 167, p. 548]; (also see incidence figures below).

The breakdown of sanitation, however, due to earthquake, civil strife, strikes, war, or the failure of authorities and citizens to plan and cooperate in the handling of the ever-expanding solid-wastes problem may provide the elements necessary to domestic and wild rodent expansion and subsequent epizootics. By the nature of civic disruptions, there may then be interference with other means of containment once the disease is under way. (See section on *Disaster* in this report.)

Once begun, human plague can spread by direct contact between humans.[167, 541, 568, 517, p. 201] American physicians "almost never see this disease" and significant human spread of the agent may occur before the nature of the disease is recognized.[165, p. 43; 167, p. 557] There would appear to be no cause for complacency with regard to plague from any source.[165, p. 42] The following quotes are found on page 529 of reference 167: "The seeming disappearance in the middle of the 19th century is merely a phase in the natural history of the disease itself. . . ." and "Knowledge acquired since 1900 leads to the conclusion that the decline of the plague at the end of the 17th century represents the operation of the great natural law of the rise and decline of epidemics."

In reference 190 (p. 1152) the following statement appears: "It might appear to some persons that too much prominence has been accorded plague and too much effort devoted to it as a public health problem in this country; but it still holds our interest, because it scaled the barrier of quarantine, because of its persistence and gradual biological and geographic spread, and because of the difficulty in eradicating it entirely in vast areas of low biological density." Another statement from the same work (p. 1149) is as follows: "When the situation is viewed in the light of the expanding areas in which plague-infected wild rodents and insect parasites have been found in recent years, the disease assumes significant public importance and becomes a problem fraught with potential danger."[190]

The ultimate threat of epidemic plague[165, 682] comes from commensal rat populations (pp. 417, 418), but the basic threat in the United States exists in the wild rodent reservoir.[165, 682, pp. 417, 418; 690, p. 1555] The first essential element for a human plague epidemic in this country — contact between commensal rats and wild rodents — is being satisfied by encroachment of urban settlements on wild areas and the use of such areas as dumps for solid wastes.[165, 166, 569] In addition, the fact that more and more Americans invade wilderness areas every year,[165] exposing themselves to sylvatic plague and producing tons of litter and garbage for which they assume no responsibility for proper disposal, is of considerable importance. This can bring about avenues of transmission made important by sheer numbers of potential victims, a growing population of wild rodents attracted to (and nourished by) such wastes, and the increasing probability of involvement of hitchhiking domestic rodents.[169, pp. 710–711]

Uncertainty as to the factors underlying the great pandemics of plague is good reason to avoid complacency in the face of its very low incidence in the United States in recent years. Only six cases were reported for 1960 to 1964,[361] eight cases during the period 1930 to 1939,[190] and 17 cases in 1932 to 1942.[162] In the period between 1900 and 1940, 499 cases with 314 deaths were recorded.[190] Between 1908 and 1965, 111 cases of human plague transmitted from wild rodents and other mammals or fleas — with 64 deaths — were reported.[690, p. 1568]

However, in a single Indian State, Uttar Pradesh, in approximately the same period (1905–1954), over 3,000,000 deaths were ascribed to plague, of which approximately 390,000 were registered in the period 1930–1954.[567] The disease almost disappeared in 1954. During the same 50 years, it showed a short-term periodicity of 5 to 6 years.[567] In Mysore, another Indian State, plague persisted, with periods of absence, into the 1960's, with progressively increasing human cases since 1960.[570] It is doubtful that the disappearance of plague in these areas could be ascribed solely to the use of pesticides or to superior sanitation; consequently the role of sanitation in the subsidence of the plague in the United States is somewhat open to question.

The main consideration is that resurgence of the disease requires a host. In India, in the two states noted above, plague was found in commensal rat fleas. In Uttar Pradesh, wild rodent fleas were also infected with *Pasteurella pestis*. In the United States, sylvatic plague is spread through the country. Presumably, recrudescence of plague among commensal rodent populations, brought to large numbers by failure to control waste,[167, p. 900] could be followed by human bubonic plague.[569, 573] One writer discounts the threat of plague epidemics in civilized countries, although he accepts the possibility that rapid spread to such areas exists.[573]

Of interest to this subject are the increases in rat populations and numbers of cases of human plague reported for the year 1966 by the World Health Organization.[692]

It would seem obvious that a population careless of its wastes in normal times would not be likely to exercise greater caution in waste disposal in the event of crises in civil life and the ensuing interruption of disposal services. Human males are especially intransigent when their "rights" are questioned to foul their homes, the surrounding landscape, and the wilderness areas with garbage and refuse. The author of this report has observed otherwise intelligent persons (men and women alike) owning wilderness cabins throwing garbage out the windows with the justifying statement that "the little animals will keep it cleaned up."

The extent to which waste and rodent control might be practiced in disaster would seem to be low, in view of the present level of hygiene actually practiced by the public (as opposed to the level provided for it by municipal sanitation services and sewerage). With the domestic rodent population out of hand and with waste accumulations attractive to wild rodents present, the threat of domestic rodent epizootics is increased. Disruption of water supply, and its interference with bathing and laundry, would encourage infestation of humans with infected rat fleas and susceptible human fleas and lice — both of which are suspect in plague transmission.[568, 573] Add to this the possible disruption of medical services and the stage is set for a human plague epidemic.

Conclusions. The rat has its greatest potential for killing humans through its role in dissemination of plague. However, this potential is difficult to assess. The adage "better safe than sorry" seems to have special significance where plague is concerned.

Recommendations. Although the epidemiology of plague sufficient to assess the total risk to human populations is known, significant exposure to populations in the United States must be through the domestic rat. General recommendations for its control are contained in the conclusions and recommendations given for rodent-borne disease as a whole.

2) Leptospirosis. General. Leptospirosis is a spirochetal disease of animals transmissible to man.[40, 517, p. 129; 682, p. 547] It is not a single disease, but a group of diseases due to a number of serologically distinct organisms.[521] The principal hosts are wild or domestic rodents and some domestic animals[72, 517, p. 130; 343, 524, 525, 382, 682, p. 547] Distribution of leptospirae is world-wide.[382]

The commonly isolated leptospirae in North America are *Leptospira icterohaemorrhagiae, Leptospira canicola, Leptospira pomona, Leptospira bataviae*, and *Leptospira autumnalis.*[531] *Leptospira pomona* is frequently the organism found in infected cattle.[522, 382]

Documented sources of human infection are rice fields,[517, p. 131] swimming 'holes',[521, pp. 142–144; 522, p. 950] sewers;[109, 517, p. 131] a number of occupations in which exposure to infected animals is intimate[526, p. 381; 517, p. 131; 343, pp. 862–863; 523, p. 22] must also be included.

Between 1949 and 1961, 483 cases of human leptospirosis in the United States were confirmed in the laboratory.[343] In 1965, however, 1,077 serum samples from routine hospital admissions showed antibody reactions to leptospiral sera in 2.6 percent of the cases.[523]

Postulation. Rat populations increase greatly if food and harborage are made available. The habits of rats expose them to leptospiral infection (sewer water, drinking water contaminated by excreters, urine of other rats) in their foraging and nesting. They come into contact with other rodents and domestic animals capable of harboring the infection. They can contaminate human food, water supply, and household articles or clothing with urine which contains leptospirae. They can contaminate water supplies from which domestic animals and pets drink; in the case of cats or dogs, which kill rats, they can serve as a source of direct contact and infection by this route. Rats thus could serve as an important link in human infection by infecting other animals having contact with humans.

Evidence. Rat populations increase greatly if given access to waste foods and waste harborage (see references, section on rodent-borne disease). Rats travel widely for food and water and to find harborage, and have migrated throughout the world.[526] They are exposed to infection by the environment contaminated by other wild or domestic hosts[382, 526, p. 378] and are themselves "chronic carriers of many types of leptospirae" and, "as a rule do not show clinically perceptible disturbance . . .".[526, p. 377; 517, p. 130; 119, 389] A lifelong carrier state is thus established. In other words, the rat population is not decreased by the disease, and their contaminating activities are not limited by the disease. Commensal rats are found to be carriers of many different leptospiral serotypes all over the world.[526, p. 377; 521, p. 141; 382, p. 473; 389, pp. 2–3, abstract]

The organisms are excreted in the urine and may contaminate the environment of man, resulting in human infection and disease.[517, p. 129; 682, p. 547] Rats are carriers and probably the chief reservoir of *Leptospira haemorrhagiae*, the agent of Weil's disease which is perhaps the most severe form of leptospirosis in man.[115, 119, 95, 526, pp. 377–378; 522, p. 950; 389, 682] Experimentally, ticks and horseflies have transmitted the disease by feeding on experimental animals[517, p. 130; 521, p. 142] and naturally infected ticks have been found on cattle in Russia.[521, p. 142] "Despite this, endemiologic evidence would indicate that arthropod vectors are not significant in the transmission of [this] infection from animal reservoirs to man."[517, pp. 130–131]

In the period 1955 to 1959, 130 cases in the United States were analyzed and 36 percent were found associated with farms or abattoirs. Some 26 percent were connected with drinking, swimming, or other immersion in presumably contaminated water; 16 percent with contact with dogs; 13 percent with rat contacts; 3 percent with wild animals; and 6 percent with other animals or environment.[521]

In the United States, according to one summary of 483 cases of leptospirosis in man in the period 1949 to 1961, a probable infecting source was ascertained in 191 cases. Sixty of these (31%) "involved contact with rats," and it was stated that ". . . infection from rats occurred in a wide variety of situations."[343, p. 863]

Decrease in cases in the period 1955 to 1965 might have been due to better sanitation and eradication measures in this country.[389]

The domestic rat may play a much more significant role as an indirect cause of human infection. It has invaded many areas to which it was not native (e.g., the Americas, Australia) and may have been instrumental in the introduction of leptospirae there. The recent introduction of *Rattus norvegicus* to some countries of the Near East, along with *Leptospira ictohaemorrhagiae*, is typical.[526] By such migrations and by virtue of its habits — especially of infesting feed lots and stalls of domestic animals — it can disseminate leptospirae to these animals. In turn, excretions of these latter animals pollute waters from which humans may become infected by working or swimming in them.[343, p. 863; 517, p. 131; 521, 522, 523, 526] More human infection is due to transmission from cattle and swine than from rats.[523]

The dog is a major source of human infection[521, p. 144; 343, p. 863] and may well be infected by the rat because of their cohabitation in the human domicile. Thus the rat may be the primary source of canine infection, and hence in some cases human infection. Some serotypes of leptospira are common to both animals, and are transmissible to man.[382, p. 473; 389, p. 6; 523, p. 21]

Conclusions. There are important gaps in a postulated chain of infection leading from the rat through domestic or other animals to man, although direct infection apparently is verified. The difficulty arising from the discovery of the same serotype in different animals is obvious — which source was the primary source? The fact that the rat is a lifetime carrier of leptospires,[517, p. 130] and that the carrier state in domestic animals is relatively short,[517, p. 130] lends importance to the rat as a reservoir. However, according to one author, outbreaks of leptospirosis are traceable to domestic animals and not to rats.[343]

In a disease as universally distributed geographically and biologically as leptospirosis, the comparative importance of the commensal rat in transmission of this disease to man is difficult to assess. The commensal rat can cause human disease, and probably domestic animal infection. The evidence supports the conclusion that it is a significant factor without whose control, or elimination, human leptospirosis cannot be eradicated,[526, p 386] especially since broad protection from immunization has not been successful.[517, p. 134; 682, p. 548]

Recommendations. The empiric involvement of the rat in this and other diseases is sufficient to warrant action without further studies, which can be left to others concerned more directly with epidemiology and medical ecology.

Mosquito-Borne Disease

GENERAL

Hunter and others[517] in 1960 listed the following diseases as being vectored by mosquitos: dengue (pp. 17, 661), encephalitis (p. 662), filariasis (p. 658), malaria (p. 317, 659), and yellow fever (pp. 20, 661).[517]

Hull[167] adds tularemia, lymphocytic choriomeningitis, and melioidosis as zoonoses in which mosquitoes can be vectors (pp. 593, 725, 846, respectively). Of these, only the mosquito-borne encephalitides and malaria are mentioned in connection with the United States.[517]

Philip was unsuccessful in transmitting Q fever by *Aedes aegypti.*[378]

Shahan and Traum (1958) list Rift Valley fever as a zoonosis transmissible to man by mosquitoes. They note, however, that this disease has not been identified outside of Africa,[57] nevertheless, there is a distinct possibility that it may be introduced to the United States[751] (p. 358; see also pp. 354–356).

Anderson,[23] among others, has referred to solid wastes in association with mosquito-borne disease. This presumptive association is due to the fact that some mosquitoes breed in small collections of water, such as may collect during rainfall in cans, automobile bodies, and other discarded potential containers. Many of these same mosquitoes, however, are known to breed in other fortuitous collections of water, such as those in hoofprints, roadside collections, irrigation water, and the like. Mosquitoes may breed in large numbers in coastal brackish waters and salt marshes and in various types of natural bodies of water, or, according to the species, in flowing streams. Thus, the contribution that might be made by solid wastes to the total threat of mosquito-borne diseases, particularly in regions already provided with other collections of water, is difficult to estimate. To produce disease, however, introduction of the disease agent into the vector population must occur. Highly complex questions of mosquito ecology and other factors must be accounted for.

POSTULATION

Solid wastes provide a source of breeding media for mosquitoes which are the vectors of disease agents pathogenic for man. These mosquitoes will oviposit in rainwater held in solid waste materials; the organic debris associated with solid wastes can serve to nourish the mosquito larvae either directly or indirectly by permitting the growth of microorganisms upon which the larvae feed. In the presence of infected hosts, the emerging adult mosquitoes will become infected and transmit the disease agent to human hosts.

EVIDENCE

Craig and Faust[319] stated in 1940 that "some mosquitoes like *Aedes aegypti, A. albopictus, Culex fatigans, C. pipiens,* and *Anopheles stephensi* are domestic in their habits, breeding in household receptacles, such as cisterns, earthenware jars, gutters, etc. Mosquitoes may fly distances of two miles or more or may be carried hundreds of miles by wind and thousands of miles in trains or airplanes. In measures directed against purely domestic Anophelines (that is *A. stephensi)*, which breed in crockery, cans, etc. around the home, careful inspection must be made by sanitary police to see that these receptacles are kept empty, covered with kerosene, or adequately screened, just as in control of *Aedes aegypti* breeding."

These authors also state, in regard to filariasis and mosquito control, that "this is essentially an urban problem. The mosquitoes are usually domestic, *Culex fatigans, C. pipiens, Aedes aegypti, A. variagatus (Anopheles stephensi)* or are found in bodies of water not far from human habitations." They state also that "man is the only definitive host of *Wuchereria bancrofti*, a cause of filariasis. Man is inoculated with mature larvae escaping into or onto human skin from the proboscis of infected mosquitoes . . . ."

Smith[332] in 1957 made the following point: "Before incriminating an arthropod as a vector of any specific disease there are certain essential requirements which must be fulfilled. In the first place, the suspected arthropod must be associated in time and place with the 'person' of the infected donor and recipient hosts. In the case of biting arthropods, this association implies that the suspected vector must bite such hosts."[332] He then pointed out that, in some cases, certain species of mosquitoes in certain locales did not bite man, whereas in other areas the same species transmitted disease. He also noted that mosquito species were not genetically uniform, and quoted Huff (1929) as showing that it was possible to breed a strain of *Culex pipiens* which, after a few generations of selection, was able to act as a host in 91 percent of the cases to a disease agent, while another line bred for insusceptibility was reduced to infection rates of the order of 7 percent. He stated further that "mosquitoes entirely unrelated geographically to [a parasite] often prove to be extremely efficient vectors . . . ."

In 1942, Hammon and others[160] reported on an epidemic of encephalitis in Washington State. They mentioned that in 1939, there had been a severe epizootic of horse encephalomyelitis involving some 500 to 600 animals, and that this was associated with about 35 human cases. In 1949 there were 86 human cases, although only about 50 horses appear to have been affected. The blood of a number of domestic and wildfowl were found to contain antibodies against either the Western or the St. Louis viruses, or both. Their own investigations revealed widespread evidence of infection among domestic fowl and domestic mammals. Only 8 percent of the sera from wild mammals possessed antibodies against encephalitis viruses. The authors concluded that "many of these birds and mammals, principally the domestic fowl, probably serve as reservoirs for the infection of mosquitoes."

Hunter and others (1960) identify two Anopheles mosquitoes in the United States as capable of transmitting malaria.[517, p. 320] These are *Anopheles freeborni* and *A. quadrimaculatus*. The former species is reported to prefer fresh, clear seepage from ditches, rice fields, edges of slow streams, and irrigation water. The latter species prefers fresh pools, ponds, lakes, lagoons, swamps, slow flowing river in dense aquatic vegetation. Both of these species enter houses and feed readily on man. *A. freeborni* is noted to be dangerous in the interior valleys of the West Coast of the United States, while *A. quadrimaculatus* is the "most important carrier in the Eastern United States." In regard to filariasis, these authors state: "Complete development of the larval forms of *W. bancrofti* has been shown to occur in over 50 species of mosquitoes including the genera *Anopheles*, *Culex*, *Aedes* and *Mansonia*. However, these mosquitoes are not all necessarily concerned with the transmission of the infection in nature. Some of the most important known vectors are *C. pipiens*, *C. quinquefasciatus (C. fatigans)*, *C. pipiens*, *Anopheles gambiae*, *A. funestus*, *A. darlingi*, *A. punctualatis*, *A. farauti*, *Aedes aegypti*, and *Aedes polynesiensis*." These authors also note that "the importance of a particular mosquito will depend to a large extent upon whether it feeds on human rather than animal blood and breeds in areas in close proximity to man."[517]

In regard to dengue, these authors state that "epidemics due to the importation of virus involving hundreds of thousands of people have occurred in areas where the mosquitos capable of transmitting dengue fever were present, notably . . . the Gulf Coast and adjacent Southern States of the United States in 1922." They list *Aedes aegypti*, *Aedes albopictus*, and *Aedes scutellaris* as the only proved vectors of the virus. Sabin, writing in this reference, says in regard to yellow fever: "Since 1948 yellow fever has advanced . . . northward through Costa Rica . . . and finally into Mexico . . . and an outbreak of yellow fever was discovered in Trinidad in 1954." Urban yellow fever is described as transmitted by *Aedes aegypti*. "This mosquito is domestic and is always found in close proximity to man, breeding in and about houses" (pp. 20–21).[517]

In 1961, Beye and others[575] reported on the hazard of importation of diseases new to the resident population of the United States by foreign migratory agricultural workers and others; for example, *W. bancrofti* (an agent of filariasis) was found among migrants in Idaho. It was noted that this agent was "once prevalent around Charleston, South Carolina, presumably brought from Africa by the 'slave migration' ", and that "the most universal mosquito vector, *Culex quinquefasciatus*, is prevalent in many parts of the United States." Also, in the Idaho survey, two cases of malaria were found. To

Beye, the finding of seven cases in California "indicates the potential hazard of this well-known parasite which was widely prevalent in the United States in the early part of the Twentieth Century. Suitable mosquito vectors are still prevalent in some parts of the United States." These authors quote Dunn and Brody, who reviewed malaria surveillance in the United States in the period of 1956 to 1957, on their documentation of three cases of primary indigenous malaria in Sacramento County, California, in 1956. Dunn and Brody stated that these cases of malaria were "possibly associated with the importation of the parasite by Mexican agricultural workers in the immediate area. *Anopheles freeborni* were present in moderate numbers." He added that "the same authors discuss four cases, three confirmed as vivax malaria in Sutter County, California. . . . *A. freeborni* were present. A farm labor camp for Mexican nationals was located about a quarter of a mile from the ranch where the cases occurred, but it was not demonstrated that anyone in the camp was the source of the infection." Beye also states that the list of potential imported diseases "could be made large . . . including such possibilities as . . . yellow fever."

In considering one aspect of solid waste disposal, the hazard of creating breeding places for mosquitoes through the use of manure lagoons was noted by Eby in 1962.[476]

Hayes and others (1962) reported on 33 confirmed human cases of Eastern encephalitis (EE) in New Jersey between August 17 and October 15, 1959.[511] They stated: "The majority of the individuals stricken resided within communities situated adjacent to woodlands and both salt- and fresh-water swamps. Arthropods were collected in the vicinity of reported human, equine, or pheasant cases. The predominant mosquito species were *Aedes sollicitans, Aedes vexans, Culisets melanura,* and *Culex salinarius.*" These investigators also stated: "All available information supports the hypothesis that (1) the swamp mosquito, *C. melanura,* served as the primary sylvan vector which carried EE virus from enzootic swamp foci to peridomestic wild and domestic avian reservoir hosts in both the epidemic (coastal) and epizootic (inland) areas; (2) *A. solicitans* served as the primary epidemic vector in the coastal area where most of the human cases occurred, obtaining its infection from the peridomestic avian reservoir hosts (including chickens) and subsequently transmitting the infection to man; (3) *A. vexans* served as the primary epizootic vector in the inland area of the equine outbreak, obtaining its infection from peridomestic avian hosts and subsequently transmitting the infection to horses; and (4) *A. vexans* may also have served as a vector for occasional human cases that occurred in the inland area." The authors feel that they have suitably accounted for this hypothesis in that they are able to reconcile it with the known habits of the vectors involved, including their feeding on both animal hosts and human beings.

Pratt and others[397] stated in 1963 that "epidemics of three types of encephalitis continue to occur in many parts of this country and are the most important mosquito-borne diseases in the United States today. Pest mosquitoes are important to human health as their continued annoyance affects physical efficiency and comfort, mental equanimity, and the enjoyment of life (Bradley, 1951)." These authors state: "Beginning in 1958, less than 100 cases of malaria have been reported each year for the entire

United States, most of them contracted overseas, with only three or four primary indigenous cases reported in 1961 and 1962." They said also that although there are 15 Anopheles species (of mosquitoes) in the United States, only two seem to be important in malaria transmission: *A. quadrimaculatus* east of the Rockies and *A. freeborni* west of the Rockies. They stated that no epidemics of yellow fever had occurred in the United States since the 1905 outbreak in New Orleans and that no major epidemic had occurred in all of the Americas since 1942. They were of the opinion that the encephalitides found in the United States were caused by different viruses transmitted normally from bird to bird, and less commonly from bird to man or his domestic animals by a number of species of mosquitoes. Suspected vectors were listed as *Culiceta melanura, Aedes sollicitans,* and *A. vexans,* and *Mansonia perturbans* in the case of Eastern encephalitis. *Culex tarsalis* was considered the most important vector of Western encephalitis.[397]

In reference 397 it is stated that "members of the Culex pipiens–quinquefasciatus complex are the chief urban vectors." These authors said that in the Tampa Bay, Florida, epidemics of 1959, 1961 and 1962, *Culex nigripalpus* was the probable vector. They stated also that "these three types of encephalitis are generally considered to be viral diseases in which birds serve as natural hosts and mosquitoes as the most important vectors. According to Hess and Holden (1958) the basic transmission cycle from bird to bird is maintained by mosquitoes with the human and horse cases considered as accidents and dead-end hosts in the chain of infection."

They say of filariasis: "Human cases of filariasis (are) not now known to be naturally acquired in the United States." They quote other workers on the temperature and humidity requirements for the successful infection by filarial worms and state: "If this is true, only a relatively small area of the United States . . . those states bordering the Gulf of Mexico, Georgia, and South Carolina . . . is favorable for the establishment of filariasis." They note as vectors *Culex quinquefasciatus, C. pipiens, Aedes polynesiensis,* and *Anopheles gambiae.* They list *Anopheles quadrimaculatus* as the most important vector of malaria in the United States, and state, "[This mosquito] breeds chiefly in permanent fresh water pools, ponds, and swamps which contain aquatic vegetation or floating debris . . . . Breeding seldom occurs in stagnant waters heavily polluted with plant or animal matter. Some of the common habitats are lime-sink ponds, pits, sloughs, bayous, sluggish streams and shallow margins and backwater areas of reservoirs and lakes. (King, Bradley and others, 1960)."[397]

These authors state that *Anopheles freeborni* (the Western malaria mosquito) "breeds in permanent or semi-permanent waters which are at least partially exposed to the sunlight, and contain vegetation or flotage. It has for the most part adapted itself to seepage, borrow pits, hoof prints, improperly irrigated fields and the edges of streams and irrigation canals." They note that the breeding places for species of Aedes are quite variable, and that "in general, they breed in temporary pools formed by rains or melting snows. Some species breed in the coastal salt marshes which are flooded at intervals by unusually high tides. Others have become adapted to irrigation practices. A few species breed in tree holes, rock pools and artificial containers." They found that the vector of urban yellow fever and dengue, *Aedes aegypti* "is thoroughly domesticated, breeding almost exclusively in artificial containers in and around human habitations." They noted that some 26 species of *Culex* had been reported in the United States. This group included several important pest species and disease vectors.

In this work it was also stated that "*Culex* mosquitoes breed in quiet waters of almost all types from that in artificial containers to large bodies of permanent waters. Water in which there is organic material including sewage is often a favorite breeding place. *Culex nigripalpus* is the proven vector of St. Louis encephalitis virus in the Tampa Bay outbreak in 1962." They found that *Culex pipiens*, the northern house mosquito, occurs throughout the northern United States and as far south as Georgia and Oklahoma, and that *Culex quinquefasciatus*, the southern house mosquito, occurs in all the southern states from coast to coast and extends northward to Nebraska, Iowa, Illinois and Ohio. They stated that the members of this species complex "are important vectors in urban epidemics of St. Louis encephalitis, particularly in the Midwest." Both species were noted "to breed prolifically in rain barrels, tanks, tin cans, and practically all types of artificial containers. These species do not migrate far except when great numbers are being produced. *Culex tarsalis* is believed to be the most important vector of encephalitis to man and horses in the western states. It is essentially a rural mosquito. The larvae utilize almost all types of water. These include canals, ditches, borrow pits, impoundments, ground pools, and hoofprints, as well as artificial containers of various types such as cans, jars, barrels, drinking troughs, ornamental ponds and catch basins." The species is noted "to fly at least ten miles, although the majority of individuals probably remain within a mile of their breeding places." In regard to the Culiseta group, "two species have been found naturally infected with encephalitis virus but their relation to the epidemiology of these diseases is not known."

Mosquito control is important in the disposal, stabilization, and treatment of solid wastes by lagooning.[744]

Thomas (1963) discussed dosage factors in the transmission of western encephalomyelitis virus, and noted that a titer in the donor required to infect the salivary glands of 7 percent of the mosquito population was $10^{-3.8}$. To infect the salivary glands of 50 percent of the mosquito population, a donor with a virus titer of $10^{-4.5}$ was required. This investigator studied the distribution of western encephalomyelitis virus in the mosquito vector, *Culex tarsalis*. He stated: "The percentage of mosquitoes infected varied directly with the degree of viremia of the donor. A single mosquito was capable of transmitting virus to a 4-day-old mouse or a 9-day-old chicken."[510]

In 1963, comments of interest to the epidemiology of malaria were made by Langmuir:[88] ". . . new criteria soon revealed that malaria had disappeared as an endemic disease from the South, probably before the DDT program had gotten underway . . . At some time between 1935 and 1945, malaria mysteriously disappeared . . . The slight rise in the morbidity curve in 1945 reflects the influx of infected veterans . . . The sharp peak in the curve in 1951 and 1952 reflects the Korean War and the occurrence of several thousand cases among veterans . . . In the past five years, the incidence of reported malaria in the whole country has remained below 100 cases a year."

Another reference to mosquito breeding in waste stabilization ponds was made by Nemerow and Bryson[68] in 1963. Such ponds are used for the reduction of solid wastes suspended in a liquid transporting medium.

In 1964, Garrison and others[150] noted that mosquitoes were attracted by wastes discarded by rural populations along the highways.

In 1964, Reeves and others,[60] reporting on 15 cases of Western encephalitis and two cases of St. Louis encephalitis in Kern County, California, in 1958, referred to high water levels following heavy rainfalls associated with an immense vector population of *Culex tarsalis*. The relatively small number of cases, in spite of this epidemic potential, was ascribed to "a combination of low temperatures in the early summer that delayed extrinsic incubation of virus in *C. tarsalis* and successful control of the vector population in the urban area by midsummer." In the same year, Aitken and others[572] reported the isolation of St. Louis encephalitis virus in Trinidad. In the years 1955 through 1962, there was a total of 19 such isolations from birds, mosquitoes, and one human being. In the same year, Dow and others[512] isolated St. Louis encephalitis virus on 22 occasions from mosquitoes taken in the Tampa Bay area of Florida during the epidemic of 1962. The mosquito chiefly involved was *Culex nigripalpus*. One other mosquito, *Culex melanconion*, was also found infected. It was stated that "these isolations indicate that infected vectors were prevalent over a vast area exposing the human population to infections throughout the region."

The worldwide nature of mosquito-borne encephalitis in humans is reviewed in the 1964 *British Medical Journal*.[514]

In the period 1960 to 1964, there were 464 cases of malaria reported in the United States. In 1964 alone, there were 102 causes.[351]

In 1965, Bond[513] reported on the 1962 epidemic of St. Louis encephalitis in Florida with the following comment: "There were 43 deaths, giving a case fatality rate of 22.2 percent. [The] virus was recovered from 4 human beings and from 42 mosquito pools, of which 40 were *Culex nigripalpus*." Of interest was the high clinical disease rate among elderly retired persons in the country. Bond stated, "Widespread viral activity in nature was demonstrated by mosquito collections and serologic findings on wild or domestic birds in each of the 4 counties. The geographic differences in human disease rates were ascribed to differing exposure factors related to wild bird density and local vegetation, and to variation in age composition, leisure time activities, or previous arbovirus experience in the human population."[513]

The multiplicity of sources of water which serve as breeding places for mosquitoes is referred to by Kimball[396] in 1965: "Breeding sources within the drainage facilities are perpetuated by accumulation of small quantities of community waste water resulting from irrigating lawns, washing sidewalks and streets, testing fire hydrants, . . . pumping out swimming pools and, in suburban areas, from irrigation of citrus groves and other agricultural operations. Natural breeding sources are creeks and ravines; natural depressions and other low areas; and salt marshes. Potential sources are ornamental pools, plastic swim pools, decorative vases, buckets, jars, cans, old tires, boats in storage, animal water, watering containers, and almost anything that will hold water for a week or more."

Adaptability of mosquitoes to environmental changes effected by man is noted in a World Health Organization Technical Report of 1965[234]: "In certain large cities . . . this threat (of mosquito-borne disease) has already become a reality, especially with regard to the rapid increase of one insect, the mosquito *Culex fatigans*, a vector of bancroftian filariasis. This mosquito has established itself in the urban environment, adapted itself to it, exploited it and turned its own peculiar characteristics to its own advantage. . . . It uses man-made breeding places to such an extent that its population density is positively correlated with that of man. In particular, these breeding places tend to be highly contaminated waters where the larvae are able to make maximum use of the organic pollution typical of an insanitary urban environment. The mosquito has the ability to shelter in houses, often in places that are difficult to reach with insecticides. It feeds on man and uses human blood for egg maturation." (See also reference 753, pp. 135–139.)

In 1965, Beaver and Orihel[742] reviewed 18 previous cases of filariasis reported in the United States, and added 21 new cases. These were due to filariae of animals. The great majority of these cases were from residents of Florida. Two cases were reported from Oklahoma and Missouri and one from Washington. In the previously reported cases, Michigan, Massachusetts, Louisiana, Texas, Wisconsin, and New York were involved. The agent of the disease, Dirofilaria, is noted by Hull[167, p. 920] to be transmitted by mosquito bite. Domestic pets, such as cats and dogs, harbor some of these forms. Beaver and Orihel state that "at present, approximately half the known cases (in the world) have occurred in the United States."

In regard to prevention of mosquito-borne disease, it has been shown that some species have become resistant to insecticides.[234]

These references, while not exhaustive, are representative of the available material on possible relationship of solid waste to mosquito-borne disease. Other references of interest to this subject are 751 (pp. 72-97), 752, and 753.

DISCUSSION AND CONCLUSIONS

The present importance and the potential for the spread to humans of mosquito-borne disease due to solid waste are impossible to state quantitatively on the basis of available information. Compared to breeding media provided by natural and man-made bodies of water, storage reservoirs, irrigation and the like, solid wastes would seem to be of little importance. However, where mosquito eradication is attempted, failure to control solid wastes as potential breeding places could well negate the program.

Under prevailing circumstances in the United States, solid waste is probably of little importance in the transmission of mosquito-borne diseases to man. Should diseases such as malaria, yellow fever, or dengue become endemic in this country, vector proximity necessary to high rates of infection between hosts and vector would be established by accumulation of solid wastes in communities or about homes. Certainly any attempt to deny the mosquito access to human habitation, whether for its potential role as a vector of disease or its role as a pest, must include control of solid wastes.

RECOMMENDATIONS

To demonstate a solid waste/mosquito-borne disease relationship, investigation would have to be done to determine (1) that infected mosquitoes are found associated with breeding media afforded by solid waste and, (2) that they can reasonably be related to human cases in the area of invasion by these mosquitoes.

The priority of this type of investigation, however, as opposed to that in other aspects of mosquito control or of solid waste disposal studies, would not appear to be very high.

Therefore, assuming that all potential mosquito breeding media are important in the spread of such diseases as viral encephalitis in the United States, the following recommendations are made:

Public education on all aspects of waste storage, including that of water collection, excessive moisture, and larval food sources conducive to mosquito breeding

Control of municipal, commercial, and industrial wastes and disposal sites in a manner preventive of mosquito breeding

Research on chemical , bological, and other controls of mosquito propagation in connection with solid waste stabilization and treatment ponds

Research on waste treatment methods preventive of mosquito propagation.

SPECIFIC DISEASE

Encephalitis (anthropod-borne). General

Anthropod-borne encephalitides are transmitted mainly by mosquitoes of various species. Western encephalitis, for example, is transmitted largely by *Culex tarsalis*, although other Culex and Aedes, Anopheles and Culiseta mosquitoes carry the disease agents.[167, p. 736]

In order to associate solid waste with this disease, linkage must be established which will take into account the ecology of the vector.

Postulation. Solid wastes of the trash variety contain materials — tin cans, old tires, plastic containers, automobile bodies and many miscellaneous items — which may catch and retain rainwater or water from other sources. Mosquitoes breed in these receptacles which are often found in close association with human habitation. The mosquitoes thus bred may feed on domestic, avian, and other hosts of the virus, which have been infected by sylvatic vectors, and in turn feed on humans to which the virus is transmitted.

Evidence. There is no direct evidence in the literature — no tracing of human encephalitis to mosquito to trash receptacle — for associating solid waste to mosquito-borne human disease. House mosquitoes, however, which breed in such containers are capable of transmitting the virus[167, pp. 733–748] and are known to feed on and become infected by animal hosts of the virus. Other species, less selective, may breed equally well in natural bodies of water, and in water made available by man through careless disposal of refuse.[167, pp. 733, 736, 741, 745, 747–748; 513, p. 399]

The epidemiologic evidence in human viral encephalitis often points to excessive breeding of mosquitoes brought on by heavy rainfall,[511, p. 119] or improper irrigation methods.

Although large mosquito populations seem to be required to result in human encephalitis, the disease has been transmitted to small animals by the bite of a single infected mosquito.[510, p. 164]

In an area of endemic encephalitis, the appearance of a human epidemic can occur through the agency of a primary sylvatic mosquito vector spreading infection to domestic animals and of a secondary mosquito transmitting the virus from these hosts to man.[511, pp. 119–120] More than one mosquito species may take part in transmissions of this type. As a final step, a mosquito is required which is capable of being infected by animal hosts and passing the virus on to man. Some of these breed in waste containers and are commonly found in human habitation (see Mosquito-borne Disease section). Little investigation has been made on the infection and infectivity of such mosquitoes actually trapped in residential areas or, specifically, in water contained in waste.

Miscellaneous Communicable Disease

FUNGUS DISEASE

General. Land fill disposal of avian manures and feathers raises the question of maintenance and spread of pathogenic soil fungi by such operations.[500, 501, 502, p. 6; 504, p. 1034–1035] Once infested, the soil may continue as a reservoir of mycotic infection for man for many years.[155] It is conceivable that disturbance of infested soils for the burial of solid waste may create a hazard.

Postulation. It is possible for pathogenic fungi to cause disease in man through the medium of solid wastes which provide the proper medium for growth of certain fungi, or, indirectly through disturbance of infested soils in preparation for solid waste disposal. It is further possible for sanitation workers handling waste contaminated by pathogenic fungi to become infected.

Evidence. In 1954, Levan[500] reported the following cases: (a) a fatal case of coccidioidomycosis in a worker after the sorting of dusty wool from an endemic area; (b) a case similar to case (a); (c) a crane operator who contracted the disease working on a project at Muroc Dry Lake; and (d) pulmonary coccidioidomycosis in a man hired to clear new land with a tractor and leveler. During this operation he was exposed to "a very high concentration of dust." The case was accepted by the insurance carrier as occupational.

Smith[332] stated in 1957, that sporotrichosis causes disease in man by infecting minor wounds. The infection was found particularly in male agricultural workers. Exposure to dust in areas of endemic coccidioidomycosis is considered especially hazardous because of the "vast numbers of dry (spores) which very readily become air-borne." Man is said to contract the infection histoplasmosis "by the inhalation of dust derived from a reservoir of fungus in soil." This author stated that "most of the evidence regarding the dispersal of fungi pathogenic for man and animal is circumstantial. The reservoir of a number of the major systemic mycoses is almost certainly soil, and for these conditions air-borne dispersal of fungal spores or of contaminated soil particles appears to be characteristic. The portal of entry of the soil-inhabiting pathogens is either the lungs (coccidioidomycosis, histoplasmosis) or a chance injury (madura foot). . .".

In 1957, Emmons[175] stated: "A factor common to many of these locations (where *Histoplasma capsulatum* was found) was the presence of fecal material of chickens or other birds, although the natural occurrence of the disease in birds has never been proved. . . . The most probable explanation for the presence of histoplasma in these sites is that the fungus grows as a saprophyte in a suitable soil and is quite independent of any animal hosts. Histoplasma may persist in soil for long periods after the environmental conditions with which its presence is usually associated has been altered. The fungus has been isolated from garden soil, from heaps of chicken droppings left in exposed sites on a hillside, and from beneath blue grass sod three years after the removal of a chicken house on the site. . . . One can conclude that fungi capable of causing human disease are frequently present in varied environments, that man must be frequently exposed to them by inhalation or trauma and that such exposures probably lead only rarely to progressive and fatal disease."[175]

In 1958, Furcolow[173] stated: ". . . histoplasmosis and coccidioidomycosis appear to be diseases of nature. The infecting organisms in these diseases grow freely in the soil and are disseminated to human beings through the medium of inhalation of the spores. . . . The present theory is that the fungus *H. capsulatum* appears to be limited to its present geographic zone by conditions of temperature and humidity. It grows in localized places where the microclimatic conditions of temperature and humidity are satisfactory and not in any generalized manner. Finally, it appears to infect people who come to these localized sites and inhale the spores. . . ."[173]

In 1958, Hosty and others[35] quoted a report of Kier and co-workers who traced histoplasmosis in an individual to the handling of chicken manure purchased by a city dweller for use as a fertilizer for flower beds. Hosty reported on three cases of histoplasmosis which involved a father and his two children, and believed that manure — probably from chickens, which had been collected by the parents — was a possible source of infection. The father's infection proved fatal.

The 1960 text of Hunter and others[517] defines coccidioidomycosis as "an acute, subacute or chronic infection of the lungs produced by *Coccidioides immitis*, acquired by inhalation . . . *Coccidioides immitis* has been recovered from the soil . . . Infection of man occurs by inhalation of dust containing the highly infectious chlamydospores . . . Primary infections have a definite seasonal incidence, occurring predominantly in the hot dusty autumn months." It was stated by Hunter that ". . . *Histoplasma capsulatum* has been isolated from the soil from numerous animals. Infection in man probably occurs by inhalation. There is no evidence of animal to man transmission."

In the book[326] edited by Sweany (1960), the following points on histoplasmosis are made: (a) In one study, the presence of the fungus in soil was associated with chicken droppings, and all the evidence pointed to "a saprophytic reservoir of the fungus in soil or organic debris from which man and animals . . . are infected," (b) Histoplasmosis should be considered a disease of nature, found in certain regions and spread to humans from some reservoir in the soil or in nature, (c) "There is no question that *H. capsulatum* is disseminated in the air. In most epidemics, . . . air-borne inhalation of spores was related to activities at the point source," (d) "Mice infected with as few as 10 spores regularly showed lesions and disease in experiments performed by Grayston and others in our laboratory," and (e) "Our present knowledge points to the soil, particularly when enriched by chicken droppings as the major source of infection."[326]

In 1961, Furcolow[505] stated: ". . . It is . . . clear that *H. capsulatum* can infect intranasally with relatively small number of spores. . . . With half a million infections and a thousand deaths a year in the United States, the importance of *H. capsulatum* to public health is self evident. The serious nature of histoplasmosis is indicated by follow-up of 100 untreated cases. With average follow-up of less than four years, one-third are dead and two-thirds of those surviving are at least 50 percent incapacitated."

In 1961, Smith and others[501] stated: "We know that in the experimental infections of animals, increasing dosage (of the spores) increases severity as indicated by deaths. . . . In one point-source epidemic we investigated, a university student dug a rattlesnake from a ground squirrel hole. Subsequently, the fungus was readily recovered from the soil. Of the seven students infected, six had symptoms and the student who wielded the shovel had the most severe illness." These authors give the annual death rate in the United States from coccidioidomycosis as ranging from 50 to 85, with an 8-year average of 64. "From 1956, when amphotericin began to be used, the number of deaths has reduced." They further state: "The saprophytism of the fungus enables it to flourish over vast areas so that eradication of it would be wholly unrealistic. Topsoil, from an Indian burial ground, where several cases originated, caused infection of several San Diegans when the soil was used for landfill. *C. immitis* was actually recovered from these fills."

In 1962, Emmons[155] stated: "Soil and humus rather than diseased man or animals are the reservoirs from which pathogenic species of Streptomyces and Nocardia entered the respiratory tract or the subcutaneous tissues of the patient. It is a fundamental concept . . . that the fungi . . . are normal components . . . of the soil or of organic debris in the soil." He also says that, once established, pathogenic fungi are capable of indefinite growth as saprophytes in suitable soil. He also refers to the association of chicken excreta and *H. capsulatum.* He states: ". . . We believe the contamination of the soil by (starlings) provides the environmental conditions suitable for the growth of this pathogenic fungus." He adds that aspergillosis is relatively rare in man. He found that "leaves and branches of trees passed through 'chippers' yield a type of coarse mulch that provides apparently optimum conditions for growth of *A. fumigatus*" — a matter of possible interest in an association with waste. He says, however, that man appears to be highly resistant to aspergillosis.

The pathogenic fungus *Cryptococcus neoformans* was reported as being isolated from barnyard soil and found regularly in old nests and droppings under roosting sites of pigeons. The author reported that "several outbreaks of pneumonitis in men exposed to old accumulations of pigeon excreta had been recorded in medical literature. Men were exposed to dust while cleaning or demolishing old buildings which had housed pigeons for many years." Quoting *Vital Statistics of the United States*, the author states that each of three mycoses (coccidioidomycosis, histoplasmosis and cryptococcosis) kills 50 to 75 persons per year, and that "the actual numbers of diagnosed and undiagnosed fatal cases may be many times these numbers." In 1964, Harrell[89] stated: ". . . One should . . . expect to find histoplasmosis acquired by pulmonary inhalation in those individuals who might have the best opportunity for contact with soil contaminated by these sources. . . . Minor epidemics of histoplasmosis have also occurred in laborers handling . . . soil specimens. Coccodioidomycosis can be considered as occupational to some extent in those whose work takes them into the desert areas of the lower Sonoran Life Zone and most especially in the San Joaquin Valley. Sporotrichosis is almost invariably acquired by the cutaneous inoculation of the organism at the time of some form of trauma to the skin."

In 1965, Campbell[504] stated: "*Histoplasma capsulatum* is not diffusively distributed in soil even in those states or regions in which it is known to be most highly endemic. To determine whether *H. capsulatum* merits consideration as a potential cause of infection in any region or community in which it has not yet been found, a more refined tool than the skin test survey is required. This is the search for foci in the immediate area. These foci are protected micropockets of soils which are heavily fertilized with an accumulation of either the excreta and feathers of wild or domestic avian or the guano of bats or both. Such micro-environments are found in . . . deserted buildings . . . town and city parks [or] any protective area where birds or bats habitually congregate to nest, roost, or hibernate. Outbreaks of histoplasmosis in Mexico after manipulation of soil indicate further that in some areas the endemic areas of histoplasmosis and coccidioidomycosis overlap." This author implicates chicken feathers as an actual agent of transmission of the organism from focus to focus. The bat is also implicated as a source of the agent.[504]

In 1965, Furcolow[502] stated: "Histoplasmosis and the other pulmonary mycoses, of which the most important are coccidioidomycosis, blastomycosis and cryptococcosis, are environmental diseases acquired by the inhalation of the agent from the atmosphere into the lungs. These fungi grow in the soil and are inhaled after the soil is stirred by some activity which creates an aerosol. In general, these organisms are small, 5 microns in size, or less, and thus readily penetrate the lungs and are retained there." He states it has been estimated that "in the U. S. alone, 30 million people have been infected with this fungus disease [histoplasmosis], and that half a million a year acquire the infection. . . . One of the most important [environmental conditions] is enrichment of the soil with bird manure. The bird species which may have been involved in enriching the soil have been extended to include not only chickens but other avians, such as starlings, grackles, blackbirds, oil birds and pigeons. . . . It is thus clear that the essential element in the soil necessary for the growth of the organism is some extract of bird manure."[502]

This author speaks of two methods of control for the disease (namely, vaccination and eradication of the fungus from its natural sources). He states: " . . . the eradication of the organism . . . is extremely difficult, if at all possible." He then makes the following statement of importance to solid waste landfill operations: "The method of covering infected with supposedly uninfected soil has been suggested . . . but, in our experience, has been only a temporary value, since positive cultures eventually reappear, probably by the growth of the organism through the covering soil with the passage of time." He refers then to very short exposure periods resulting in clinical disease. With respect to solid waste disposal, he states: ". . . a wooded area along a creek . . . was cleared by bulldozer operations for the construction of a bridge. . . . The bulldozer operator became ill and subsequently died. A number of other persons were made ill and a clinical diagnosis of histoplasmosis was made in 29 persons. Consideration of all the facts revealed by the studies made it quite apparent that there had been a widespread aerosol created by the felling of the trees and the activities of the bulldozer during the hot dry summer period in which the clearing operation took place."[502]

In 1965, Dodge *et al.*[503] reported an outbreak of histoplasmosis among school children who had played in a school yard in which the ground had been contaminated by bird droppings under a starling roost. These authors noted that "the absence of grass may relieve the fungus of competition for food, especially for essential elements which may be present in the bird droppings. The uses to which the school yard was put, both as a playground and parking place, helped to create much dust with its load of spores and put a high concentration of humans at risk of breathing the dust and spores."

In 1966 it was reported[100] that micro-epidemics of sporotrichosis can occur throughout the United States. It was stated in that work that "Sphagnum moss apparently served as the vehicle for the transmission of the disease." Material already quoted above was repeated in regard for histoplasmosis and aspergillosis.

In 1964, Dixon and McCabe stated [109] that fungi potentially pathologic for man had been isolated from sewage. Because of the low morbidity generally, the authors believed that the presence of such fungi in sewage did not lead to a significant extra health hazard.

Fungi have been found in sewage sludges, but no mention was made as to whether or not they were pathogenic. [211]

Discussion. In all of the material cited, there is not a single reference to infection by fungi of sanitation workers in association with sanitary landfills, dumps, or other means of disposal of solid wastes. Neither is there any specific reference to parks or playgrounds which have been formed over sanitary landfill. However, dumping of untreated domestic avian manures in landfills, or scattering of this excrement as fertilizer in areas which later could become used for recreational purposes, provides the potential means of dissemination of the pathogenic soil fungi. In connection with the practice of covering waste with soil in the sanitary landfill, it is of interest to note that histoplasma can penetrate soil after having been buried. [502, p. 7]

The potential hazard to landfill employees is suggested by the short exposure time necessary for infection and the appearance of pulmonary infection in personnel associated with bulldozing of infested soil. [502, p. 8; 501, p. 310] The hazard from histoplasma to communities from nearby landfill operations is suggested by the evidence of widespread dissemination of the infection as the result of bulldozing and clearing operations. [502, p. 9] The use of bird manure as fertilizers in parks and playgrounds thus comes into question. [502, pp. 5–6]

The fact that no infections due to soil fungi have been reported among sanitation workers engaged in sanitary landfill operations, or in the general public using parks or playgrounds developed from sanitary landfills, is no guarantee that infection has not occurred due to pathogenic fungi resulting from the nourishment provided by organic wastes.

Until attention was drawn to histoplasmosis and the existence of widespread foci, it was thought that this disease was rare.

The extremely small dose or numbers of infectious elements of the fungi necessary to cause disease [505, p. 304] suggests a definite potential for infection where avian manures are disposed. In addition, it would appear that the pulmonary deposition of the agent of histoplasmosis is not necessary, at least in mice, for the development of the disease. [505, p. 304]

A significant factor relating to the dissemination of pathogenic soil fungi in the operation of sanitary landfills is the disturbance of the soil of the site in preparing trenches or in moving debris and the frequently attendant dust raised by both trenching and covering. Air-borne pathogens are thereby potentially introduced to unprotected landfill workers.

It is interesting to speculate that if the bat is actually a carrier of *Histoplasma capsulatum*,[504, p. 1335] and hence a disseminator for the development of foci, the proper control of garbage could act to reduce the number of insects which provide the basic food for this animal — an animal that is also a carrier of rabies and other zoonoses.

Conclusions. Present data do not permit any estimate of the possible hazard of pathogenic soil fungi in association with solid wastes. On the one hand, the earlier consideration of histoplasmosis as an uncommon disease, and its ultimate revelation as a very widespread infection, does not permit an offhand dismissal of a significant relationship between the diseases caused by these fungi and solid wastes handling. On the other hand, the importance of the disease from the standpoint of morbidity and mortality should not be over-estimated pending further research on its epidemiology and prevalence among specific populations known to be directly or indirectly associated with solid waste disposal.

Recommendations. In considering disposal methods, application of those forms of treatment which destroy pathogens would seem highly preferable to those which merely cause reduction in their numbers, or no reduction at all.

Since the fate of the pathogenic soil fungi in sanitary landfills is not known, both current landfill operations and former landfill sites converted to recreational areas should be studied. The suppression or multiplication of the agents by landfill methods would have to be known if the potential for infection is to be estimated.

Epidemiologic studies of histoplasmosis in the general population should include consideration of solid waste disposal sites in tracing the source of infections.

Sanitation workers should be screened to discover the prevalence of the infection among them as opposed to that of the regional population in general. A careful determination of the presence of foci in the areas worked by sanitation workers should also be carried out. [One author feels that population surveys alone are not dependable, at least in the case of histoplasmosis.[504]]

The use of sanitary landfills as parks and playgrounds perhaps should be prefaced by investigation of the potentialities for culture and dissemination of pathogenic fungi.

Fertilizers prepared from chicken manure particularly should be examined for their ability to create a favorable environment for the implantation and multiplication of fungi. Similar investigations of other wastes may be required to determine if they support other types of pathogenic fungi, or help to create favorable environment for their growth. Composts and sludges used as soil conditioners also should be investigated.[502, pp. 5, 6; 503, p. 1209]

Consideration of pathogenic soil fungi would thus seem appropriate in selecting sites for sanitary landfills and in the choice of the material to be disposed and used for cover in the fills. A question for the future, in this regard, might be the following: Will landfills in which large amounts of avian manure are disposed become eradicable reservoirs of *Histoplasma capsulatum?* [155] The general question, of course, is this: Can waste create the environmental conditions necessary to propagation of pathogenic soil fungi? The concern here, of course, is that, once infested, the soil may continue as a reservoir of mycotic infection for man for many years. [15] It is therefore suggested that sanitary landfills used as parks or other recreational purposes be maintained under surveillance, at least until it is established that no risk exists of infection by fungi.

ANTHRAX

General. It has been suggested that anthrax may arise from contact with animal wastes. [23]

Hull [167] states that anthrax is primarily a disease of animals; that secondarily, man is infected from an animal, either by direct or indirect means (page 82); and that it occurs in all parts of the world (p. 88). He states that "practically all animals are in some degree susceptible to anthrax. Cattle, horses, mules, sheep, goats and the wild herbivores are most commonly affected. . . . Although anthrax in livestock in the United States is principally confined to cattle, outbreaks in horses, mules, sheep, and swine are also encountered. Infection in livestock usually is a result of grazing on infected pastureland rather than by contact. Infection may also be caused by contaminated fodder or artificial feed-stuffs, such as bonemeal, fish meal or oilcake and tankage; by drinking from contaminated pools; or by the bites of contaminated flies (pp. 95–96) . . . . To prevent the introduction of anthrax into the United States, Federal regulations governing the importation of bones, or bonemeal for use in animal feeds or fertilizer are enforced by the Department of Agriculture." [167]

Postulation. Anthrax may be transmitted by indirect modes involving wastes. The disease agents might be disseminated in animal excreta, by flies as universal carriers of disease agents, in tissues of dead animals, or in wastes from animal product manufacturers such as the leather industry.

Evidence. In 1907, Buchanan [360] in investigating the carriage of infection by flies, found that "a very profuse growth of *Bacillus anthracis*" resulted when flies contaminated with the bacillus were permitted to walk on agar surfaces. In 1913, Graham-Smith [121] stated in regard to anthrax," . . . under suitable conditions, which are not frequently fulfilled, the bacillus may be distributed by flies in many ways, though no definite evidence of infection either in men or animals has yet been obtained" (p. 186).

In 1914, Mitzmain [359] found that biting flies could not play a role in the transmission of anthrax "until the peripheral circulation becomes invaded with tremendous numbers of the anthrax bacterium." The stablefly, *Stomoxys calcitrans,* was found to transmit anthrax when permitted to feed on animals recently dead of the disease (that is, within the first few seconds or minutes following death). Guinea pigs exposed to the bite of the flies died and "typical pictures of anthrax infection were presented at the necropsy. . . . Pure cultures were obtained from the spleens of the dead animals." This investigator also noted that "typical organisms were seen in the feces of horseflies at various intervals up to 48 hours from the time the infected animal was bitten. The feces of the stablefly were likewise found to be infected up to 24 hours after obtaining blood from a sick animal." [359]

West [90] reported in 1951, that flies were capable of transmitting anthrax, but gave no factual data of an epidemiologic nature. Similarly, Chandler implicates the Diptera as a mechanical conveyor of anthrax (p. 663). In 1961, Herms [195] stated: "It has been amply proved that coprophagous fly larvae [maggots] . . . may transfer bacteria . . . through the pupal stage to the mature flies. In this manner, the infection of anthrax may be disseminated by flesh flies, bred in carcasses of animals that have died of this disease" (p. 22).

In 1957, Smith [332] stated: "Spread of the disease from animal to animal by direct contact rarely occurs; there is some medium through which infection most usually occurs. Infection takes place by the alimentary tract and the bacilli are excreted by the urine, faeces, and possibly the milk during the latter stages of infection. Bloodstained infected fluid exudes from all orifices of the body at death, and the immediate surroundings may be heavily contaminated. The bacilli do not sporulate in the unopened carcass, but when the bacteria are exposed to atmospheric conditions spores are produced and these are resistant and may remain alive in or on infected soil for many, many years. Animals may therefore be infected by feeding on contaminated pasture land and the greater incidence of infection in swampy areas and during the summer months is associated with the development of the spores and the multiplication of the bacteria on the decaying vegetable matter in these areas. In Great Britain, outbreaks are associated with feeding imported infected foods. The disease is therefore not only more common during winter months when artificial foodstuffs are used but is clearly more frequently observed amongst cattle which are fed on imported foodstuffs than amongst those cattle and sheep which are fed in a different manner." [332] In speaking of this disease in 1958, Shahan and Traum [57] state: "In 1952 anthrax occurred extensively in animals in some areas of the United States, particularly in the Middle Western states. It is believed that at least one outbreak was caused by the feeding of imported bonemeal containing anthrax spores."

In 1962, Klein *et al.* quoted another author, ". . . Tannery wastes may contain the very resistant spores of anthrax bacilli derived from hides." In 1965, Okum and others [70] quoted Jansky to the effect that he was able to recover hair and flesh from tannery wastes.

In regard to incidence of the disease, both in animals and man, it has been stated that in Iran during 1945, one million of a total of 15 million sheep died of anthrax. [682] In the same work it is stated: "In the United States scattered outbreaks are widespread, but the disease is not a continuing problem except in certain areas. During the period 1945–1954, 3,447 outbreaks were reported from 39 states with loss of 17,600 head livestock." In the United States, approximately 50 cases of human cutaneous anthrax were said to be reported annually, with the probability that additional unreported cases occur.

It was further stated: "Man is infected by contact with infected animals (agricultural anthrax) or contaminated animal products (industrial anthrax). Agricultural anthrax occurs in farmers, veterinarians, and slaughterhouse workers. Industrial anthrax occurs in persons whose work brings them in contact with contaminated hair, wool or hides although other products have been responsible for scattered outbreaks. Dock workers are occasionally infected while handling contaminated hair and hides."[682]

In its report of vector-borne diseases in the United States, the Public Health Service affirmed that there were 54 cases of anthrax during the period of 1960 to 1964. In 1964, five cases of vector-borne anthrax were reported. Hull stated that the yearly incidence of anthrax in man for the period 1945 to 1960 was less than 70 cases. This author reported that anthrax fatalities occurred in tannery industries in both Germany and the United States in the period 1927 to 1932. This author quoted the Smith reports for the period 1919 to 1943 in which human cases were derived from "hides and skins of cattle, goats, horses, and mules, horse-hair, imported wool, blood meal, and fertilizer." Numerous cases of occupational origin (none involving sanitation workers) were mentioned. Suggested links, were noted, however; it was found that "other cases occurred in rendering plants and from the use of fertilizers, the material in one instance being wool waste." Economic pressures often result in the conversion of former wastes into by-products, as in the case of food or other processing activities. Whereas bones formerly were disposed of as waste, they are now converted into fertilizers and bonemeal. A number of fatal cases were reported by Hull to have been the result of infection through bones, bonemeal, or fertilizer.[167, p. 102] Hull also mentions infections conveyed by biting flies (p. 103). Since dead animals are a major waste source, the following quote from Hull is of interest: "Bites from pets which have recently fed on the carcasses of animals dead of anthrax have been known to cause anthrax infection" (p. 103). Also of interest is the statement: "Contact may be more remote, as handling hides which have been shipped long distances. . . . the contact may be very remote, as a man . . . becoming infected by a shaving brush made from hair of an infected animal in China" (p. 104).

Hull also reports of a case of anthrax in a New York laborer cleaning out a sewer (p. 107).

In 1960, Reed and McMillan[531] quote a World Health Organization committee report on anthrax to the effect that, while there are approximately 9,000 cases of human anthrax reported annually, the actual incidence (due to underreporting) may be as much as 90,000. These authors also note that tannery wastes may serve as a potential source of contamination, as may artificial feeding stuffs such as bonemeal.

In 1964, Meyer[581] stated that "anthrax is an occupational infection of . . . handlers of hides or fibers". He also made the following statements: "Asia has been distributing contaminated animal by-products such as hair, wool, and skin, and this has reverberated in industrial plants and in agriculture elsewhere through use of unsterilized boneflour and bone, hoof or meatmeal as artificial feeds on farms. The U. S. Department of Agriculture has established new requirements that have eliminated the risk from that source."

Discussion. Present laws and agricultural practices in the United States have resulted in a very small number of cases of human anthrax in recent years. The present total risk of anthrax as a possible disease of sanitation workers is implied in the one quoted case involving a sewer worker. However, as pointed out by Hull[167] (p. 106, p. 111), the hazard from dead animals which have succumbed to this disease is extremely high. In fact, the extreme virulence of anthrax, as implied in the cases quoted above, could easily result in the spread of infection to sanitary workers and others handling waste animal products should anthrax become established on a broader scale in this country among the animal population.

Conclusions. The present level of anthrax in animals and humans in the United States is low enough so as to present little risk to American sanitation workers. To the extent that the laws of the United States are not carried out with respect to introduction of contaminated materials from foreign countries, there will be a certain risk. This risk of becoming infected will extend to persons both within and without the animal products industry through the handling of waste materials. The risk is especially high among persons attempting to dispose of the bodies of animals dead of the disease, and very special precautions must be taken in the event of suspected animal anthrax (Hull, p. 111).[167]

Recommendations. The virulence and infectivity of anthrax is well known, as are other epidemiologic factors of concern to waste handling. The continued education of the animal products industry, and the enforcement of existing laws along with inspection, bacteriological testing of imported animal products, and testing by the industry of the manufactured product should suffice to control anthrax. There is no further requirement at the present level of anthrax in the United States.

TRICHINOSIS

General. Trichinosis in the United States is mainly transmitted to humans by ingestion of pork containing viable larvae of the nematode *Trichinella spiralis*. The disease is widely spread among carnivores and other animals of all types. It also is found in aquatic mammals.[507, p. 159; 508, p. 314] The eating of such animals has led to trichinosis in man.[167, p. 438]

Postulation. The epidemiology regarding the usual transmission of trichinosis to man requires no postulation as far as solid waste is concerned.

Evidence. It is known that the feeding of raw garbage to hogs is the major factor in the completion of the hog/human cycle.[507, p. 160; 167, pp. 437, 438, 440; 508, p. 313] Larvae contained in raw or improperly cooked pork scraps are consumed by garbage-fed pigs and complete their encystment in the animal, whose meat is then eaten by the human. If the larvae are not killed prior to consumption of the meat, the worm completes its human cycle.

The evidence is twofold: first, the reduction in feeding of raw garbage to hogs has resulted in a marked reduction in swine infections, [506, p. 872] and, second, grain-fed animals only rarely contain trichina. [508, p. 316]

The decrease in prevalence and degree of infection in man and pigs followed "enactment and enforcement of regulations prohibiting the use of uncooked garbage in swine feed." [506, p. 872] It is ironic to find that these controls were instituted to control virus diseases of swine and not trichiniasis in man. [507, p. 160] However, with only 2 percent of slaughtered hogs fed on garbage, "the density of infection in them was sufficiently great to contribute significantly to infection in man." [506, p. 872] The continued feeding of raw garbage to hogs still accounts for localized trichinosis, [507, p. 161; 508, p. 314] and for infested meat from slaughterhouses buying hogs from many sources. [509, p. 237]

Rats associated with dumps have been found infected, but the relationship of rats to swine and human infection is not understood. [508, pp. 317–318; 506, p. 872]

Discussion, Conclusions and Recommendations. The epidemiology and means for prevention of trichinosis in humans is well understood. No further research on the disease in regard to solid wastes is required.

TRYPANOSOMIASES

General. Chagas' disease in America, caused by *Trypanosoma cruzi*, is transmitted by reduviid bugs. It is a typical zoonosis, and if man enters a natural focus he may accidentally acquire the disease when attacked by the wild vector.

Raccoons can be spontaneously infected with *T. cruzi.* In the United States, raccoons raid human habitats in search of food, especially if reclamation of land by man destroys the natural habitat (Hoare, pp. 288–291). [555] The attraction of house, garbage, garbage dumps and indiscriminate litter of food, can — as in the case of rats — support a raccoon population close to human habitat. In Chagas' disease, the vector has adapted itself to the human domicile and feeds on human blood (p. 291). [555]

Such adaptation is representative of the probable evolution of many diseases of man, and there is certainly no reason to believe that such evolution has stopped. There is no guarantee that new adaptations will not occur through man's activities in disturbing the natural vector/feral animal relationship (pp. 285–286, 291). [555] Thus man's failure to dispose properly of his wastes can contribute to increasing the number of disease agents to which he is susceptible.

In the United States, clinical Chagas' disease, in spite of natural foci of infection, is very rare. This may be due to a number of factors, including variation in the habits of the local vector (p. 292). [555]

Discussion and Conclusions. This material has been presented only to suggest the extent to which solid wastes can provide a potential threat to man through adaptive or evolutional mechanisms. Ecology is not a static phenomenon; on the contrary, it is extremely dynamic. Evolutionary changes can occur in very short periods — witness the Australian experience with rabbits and myxomatosis.

The following references are given as evidence of adaptive and mutational changes:

560 (pp. 340, 341, 355)
558 (pp. 363, 366, 368)
57 (pp. 614–615)
555 (pp. 285–287)
581
583 (pp. 468-469)
154
155
156
162
163
164
168 (p. 2 of the abstract)
174
195
196
234
313
319
331 (p. 2 of the abstract)
332 (pp. 5–6 of the abstract)
344 (p. 2 of the abstract)
391
587
591 (p. 1291)
592 (p. 287)
519 (p. 2 of the abstract)
552 (p. 2 of the abstract)
561
567
569 (p. 2 of the abstract)

Recommendations. Those concerned with the public health aspects of solid waste should give attention to the adaptivity of disease agents and animal hosts toward expanding the number of infections to which man is subject or increasing the frequency or degree to which he becomes infected.

CROP DISEASES

Although a number of disease agents associated with solid wastes do not attack man, they may still have an adverse effect on his health or well-being. For example, certain disease agents attack the animals or crops which supply man's food. The former have already been discussed in brief.

The intent here is to note, but not document in detail, the fact that there are crop diseases for which crop wastes, or insects associated with waste, carry the disease agents that damage or destroy the plant food products of man and his domestic animals.

Due to their enormous reproductive capacity, even small numbers of insects finding harbor in waste can infect entire crops and herds the following season. However, critical reduction in the wintering population can reduce the extent of insect damage.[86]

The following quotations pertinent to this relation are taken from Metcalf and Flint: [86] "Many crop insects hide under surface trash such as boards, boxes, sacks, brush heaps, stone piles, dense grass, fallen leaves and other dead vegetation" (p. 410). "Stored grain pests can maintain themselves in waste grains and screenings." (p. 933). "Bean vines and other bean plant refuse help sustain the bean weevil" (p. 938).

Metcalf and Flint found that plant wastes harbor insect pests in various stages of development (p. 932). Since fruit flies deposit their eggs in the rind, they suggested the following control measure: "In infested areas fallen fruit should be collected daily and buried to a depth of several feet or burned or cooked to destroy the larvae" (p. 814). To control the peach bark beetle they advised that "all peach prunings and dying or diseased trees . . . be removed and burned during the winter" (p. 756).[86]

These comments are typical of those to be found in texts on insect pests and plant diseases dealing with crop wastes.

E. injuries and safety considerations

General

Solid waste disposal comprises a number of occupations, including operation of trucks, incinerators, bulldozers and other equipment, as well as various kinds of manual work.

Solid wastes often are handled, either purposefully or accidentally, by householders and their families. Exposure to hazards depends on the particular work performed, the nature of the material handled, and the extent of protection provided and used — including safety practices as well as equipment.

Postulation

In view of the spread of sources and kinds of solid wastes, as well as the variation in methods of disposal, *a priori* statements on the hazards involved in their handling can only reflect a feeling that risks are undoubtedly incurred. Some of these, other than mechanical hazards, have been discussed elsewhere in this report.

The possible exposure, for example, of sanitation workers to biologic pathogens when handling sludge can be inferred by the probable presence of disease agents in some of these residues.[16, 215, 658]

Evidence

SANITATION WORKERS

Sliepcevich[134] noted high-frequency rates (69.2) of injuries in "uniformed sanitationmen" in New York City. She noted comparative excesses (relative to the national average) in arthritis and in muscle and tendon disease — especially of the back — among refuse collectors. Skin disease was not found to be excessive.

Except for the negative skin lesion correlation, this report does not refer to disease or injuries one might postulate as peculiar to solid wastes (that is, rat bites, enteric diseases, chemical intoxication, and the like).

3

One author speaks of the kinds of hazards present in refuse collection but offers no data except the fact that accident rates were reduced following the institution of a safety program.[447] Similar reports dealt with waste incineration hazards[448] and street sanitation.[449] The danger of soiled clothing to refuse collectors (skin rashes, irritations) and potential infection of minor injuries from such clothing is described.[451]

Van Kleeck[445] states that sanitary landfills are hazardous to employees and the public. Vehicular operation, dust, fires, contamination, explosives and mechanical hazards are listed as the possible sources of risk. No frequency or severity data are given.

Wolfe quotes Mail as stating that the risk of poisoning by pesticide wastes among refuse landfill employees is minimal.[15]

The American Public Works Association Committee on Refuse Collection and Disposal made the following statement in its 1958 edition of *Refuse Collection Practice*[273]: "While not a hazardous occupation, refuse collection has numerous perils and dangers for workmen who are not alert or well trained." (p.325)

The Committee also pointed out the dangers to workmen involved in refuse collection such as the lifting of heavy loads, the handling of defective containers, and the incurring of cuts and abrasions in handling the actual refuse.

Gotaas[314] refers to special precautions to be taken to protect compost plant workers when night-soil or sewage sludge is added in the operation, but gives no information on histories of infection associated with such work.

Hand-forking to remove bed-dried digested sludge is mentioned, but no reference is made to any hazard from such a practice.[215]

One report[211] discusses the health hazards of sewage treatment workers, but does not implicate sewage sludge in this regard. Other reports[71, 73, 109, 222, 254, 445] deal with sewer and sewage plant working hazards, but do not refer to any solid wastes in that connection, except to say that sludge gas collection is hazardous. Another report on sewage plant worker hazards states that leptospirosis and infectious hepatitis may be occupational diseases among employees.[109] Personnel working with sewage solids or sludges are not mentioned.

Demolition is implicated in cryptococcosis infection among building demolition workers, but involvement of persons acting as "waste handlers" is not mentioned.[155]

THE PUBLIC

One report[15] refers to pesticide fatality or intoxication in children playing with discarded containers. Another lists the possible hazards to the public at the site of refuse landfills.[445]

Safety Survey

Since the literature contained very little on occupational hazards of sanitation workers in general, and still less on those relating only to solid waste, a questionnaire based in part on information given in reference 273 was sent to 500 municipalities, 325 industries, 10 industrial insurance companies, and 50 state health departments.

MUNICIPAL SANITATION WORKERS

Questionnaires. Questionnaires were sent to all cities in excess of 50,000 population, in addition to a random selection of cities having populations between 20,000 and 50,000. Of the 500 questionnaires distributed, only 97 (i.e., 19.3%) responded. Included in the 97 responses were 28 answers containing no information other than that solid wastes were collected and disposed of by private contractors. A few responders in this group indicated they were forwarding the questionnaire to the contractor, but no information resulted. An additional 33 questionnaires contained only partial information (i.e., undocumented estimates of injury and illness; data on number of employees, basic work week, tonnage, etc., with no injury or illness figures, or the reverse). In the final analysis, 36 responses (7.2%) contained sufficient data for review and comparison. Almost 93 percent of the 500 municipalities approached were either not capable of providing the information requested or did not consider the subject important enough to be considered.

Injury Data. The information requested consisted of the number and types of injury and illness recorded over as long a period as available, the number of tons of refuse collected annually, the basic work week, the type of collection vehicles used, and whether or not the organization operated a training program. After receipt of the information, three additional variables were added from data contained in the APWA *Refuse Collection Practice.*[273] These variables were (a) maximum container size, (b) whether or not bulk material was picked up loose or tied, and (c) the requirement for furnishing of uniforms for the collection workers. Table 1 shows the variables considered in the analysis. A series of regression analyses were run in an attempt to discover any possible relationships between the various controllable variables and the dependent total injury frequency rate (not the disabling injury frequency rates). Total injury frequency rates were 282, and disabling injury frequency rates were 156.

The disabling injury frequency rates assume normal two-week vacations only, and do not account for sick leave or holidays since such information was not available. The results would thus provide a comparably more conservative rate than that obtained by the Bureau of Labor Statistics.

Statistical Analysis. A series of regression analyses were run in an attempt to determine factors affecting sanitation workers' injury frequency.

The first step in the analysis was to consider that subset of the data wherein complete information was available for all variables. There were 32 observations of this type. A multiple regression equation of the form:

$$Y = b_0 + b_1X_1 + b_2X_2 + b_3X_3 + b_4X_4 + b_5X_5 + b_6X_6 + b_7X_7 \tag{1}$$

TABLE 1

EXPLANATION OF TERMS USED IN TABLE 2

Y = Injury frequency,[a] number/10^6 man hours

X_1 = Tons/worker

X_2 = Basic work week, hours

X_3 = Type vehicle
  CT[b] — 3.0
  EN[c] — 2.0
  N[d] — 1.0

X_4 = Training program
  Yes — 2.0
  No — 1.0

X_5 = Maximum container size, gallons

X_6 = Bulk material
  Tied — 2.0
  Loose — 1.0

X_7 = Uniforms required
  Yes — 2.0
  No — 1.0

[a] Injury frequency here refers to all injuries rather than disabling injuries.

[b] CT = Compactor truck.

[c] EN = Enclosed, non-compactor truck.

[d] N = Open truck.

was estimated. The set of b coefficients was estimated by least squares. Notations for the dependent and independent variables are defined in Table 1. Basic data are shown in Table 2.

The fitted equation did not explain a large amount of the observed variation in injury frequency. The multiple correlation coefficient was 0.56 and the accompanying coefficient of determination was 0.32 (i.e., the equation accounted for only 32% of the variation). According to the results of the analysis, however, none of the partial regression coefficients were individually significant.

This type of analysis is affected by correlation of the independent variables. Table 3 shows a matrix of simple correlation coefficients which was extracted from the multiple regression computer printout. The matrix is symmetric around the diagonal, requiring only the upper half to be shown. The circled coefficients are those which were significantly large. A coefficient larger than 0.35 would be expected to occur less than one time in twenty, and a value of 0.45 or greater would occur one time in a hundred by chance.

Table 3 indicates that the correlations of tons per worker, work week, type of vehicle, type of material, and uniform required are not significant. However, the correlation of 0.33 for training programs and 0.37 for large container size suggest a possible relationship. The implicated association between tons per worker and container size deserves further consideration. It is possible that some cities estimate their total tonnage on the basis of container size as opposed to direct weighing of the disposed material. The correlation between shorter work weeks and training programs could be meaningful since it might imply recognition by some authorities that excessive hours worked can lead to over-tiredness and consequent injuries. It might also, however, reflect labor union demands. At any rate, this factor might lend credence to the rather insignificant correlation between work week and injury frequency.

TABLE 2

SANITATION WORKERS' INJURY–FREQUENCY DATA *

Y	X_1	X_2	X_3	X_4	X_5	X_6	X_7
436	–	40.0	3.0	1.0	–	–	–
209	–	40.0	3.0	1.0	–	–	–
345	–	40.0	3.0	1.0	–	–	–
119	–	40.0	3.0	1.0	–	–	–
134	580	40.0	2.8	1.0	–	–	–
201	1340	44.0	3.0	2.0	–	–	–
240	585	40.0	2.9	2.0	40	1.0	1.0
430	585	40.0	2.9	2.0	40	1.0	1.0
381	585	40.0	2.9	2.0	40	1.0	1.0
530	585	40.0	2.9	2.0	40	1.0	1.0
331	730	40.0	2.8	2.0	32	2.0	2.0
399	730	40.0	2.8	2.0	32	2.0	2.0
78	2000	48.0	2.5	1.0	30	2.0	1.0
68	740	40.0	1.5	2.0	–	–	–
455	450	46.0	1.5	2.0	30	1.0	1.0
364	665	40.0	–	2.0	–	–	–
217	610	40.0	–	1.0	–	–	–
348	610	40.0	–	1.0	–	–	–
174	610	40.0	–	1.0	–	–	–
261	610	40.0	–	1.0	–	–	–
326	610	40.0	–	1.0	–	–	–
174	610	40.0	–	1.0	–	–	–
282	610	40.0	–	1.0	–	–	–
348	610	40.0	–	1.0	–	–	–
239	610	40.0	–	1.0	–	–	–
100	–	40.0	3.0	2.0	30	1.0	2.0
128	–	40.0	3.0	2.0	30	1.0	2.0
120	–	40.0	3.0	2.0	30	1.0	2.0
500	395	40.0	3.0	1.0	25	2.0	1.0
355	1310	40.0	2.6	2.0	60	1.0	1.0
258	1370	40.0	2.6	2.0	60	1.0	1.0
375	1430	40.0	2.6	2.0	60	1.0	1.0
411	1490	40.0	2.6	2.0	60	1.0	1.0
460	1540	40.0	2.6	2.0	60	1.0	1.0
221	565	40.0	3.0	2.0	50	2.0	2.0
142	380	40.0	3.0	1.0	32	2.0	1.0
167	353	40.0	2.0	1.0	30	2.0	1.0
608	757	40.0	–	2.0	–	–	–
136	605	40.0	2.8	1.0	20	1.0	1.0
105	217	40.0	3.0	2.0	20	1.0	1.0

* Code to letters is given in Table 1.

TABLE 2 *(cont.)* *

Y	X_1	X_2	X_3	X_4	X_5	X_6	X_7
100	218	40.0	3.0	2.0	20	1.0	1.0
104	222	40.0	3.0	2.0	20	1.0	1.0
116	226	40.0	3.0	2.0	20	1.0	1.0
122	236	40.0	3.0	2.0	20	1.0	1.0
270	1010	40.0	3.0	2.0	–	–	–
595	1640	45.0	–	1.0	–	–	–
152	1260	45.0	3.0	1.0	30	2.0	1.0
159	542	40.0	3.0	1.0	–	–	–
210	500	40.0	3.0	1.0	60	1.0	1.0
612	1420	48.0	3.0	2.0	–	–	–
282	600	40.0	2.8	2.0	–	–	–
433	1390	40.0	3.0	1.0	–	–	–
183	1220	40.0	–	2.0	–	–	–
315	480	48.0	–	1.0	–	–	–
365	480	48.0	–	1.0	–	–	–
455	480	48.0	–	1.0	–	–	–
425	480	48.0	–	1.0	–	–	–
310	480	48.0	–	1.0	–	–	–
378	820	40.0	–	2.0	–	–	–
275	755	40.0	1.5	2.0	30	2.0	1.0
335	755	40.0	1.5	2.0	30	2.0	1.0
305	755	40.0	1.5	2.0	30	2.0	1.0
360	755	40.0	1.5	2.0	30	2.0	1.0
300	755	40.0	1.5	2.0	30	2.0	1.0
132	480	44.0	2.7	1.0	60	1.0	1.0
345	200	40.0	3.0	1.0	60	1.0	1.0

* Code to letters is given in Table 1.

TABLE 3

CORRELATION COEFFICIENTS, SANITATION WORKERS' INJURY FREQUENCY

		Injury frequency	Tons/ Worker	Work week	Type vehicle	Training program	Container size	Material type	Uniform required
Y	Injury frequency	1.00	0.21	–0.20	–0.24	0.33	0.37	–0.01	0.10
X_1	Tons/Worker	–	1.00	0.36	–0.17	0.05	0.45	0.12	–0.03
X_2	Work week	–	–	1.00	–0.12	–0.37	–0.05	0.12	–0.12
X_3	Type vehicle	–	–	–	1.00	–0.24	0.12	–0.44	0.17
X_4	Training program	–	–	–	–	1.00	–0.04	0.19	0.20
X_5	Container size	–	–	–	–	–	1.00	–0.34	0.01
X_6	Material type	–	–	–	–	–	–	1.00	0.39
X_7	Uniforms required	–	–	–	–	–	–	–	1.00

The two remaining circled coefficients are based on qualitative variables. Thus the significance tests of the product–moment correlation coefficient may tend to be biased. The relatively high correlation between vehicle type and material type possibly reflects regulations necessitated by type of vehicles used. The possible relationship between material type and uniforms defies explanation.

The next step in the analysis consisted of fitting a series of simple linear regressions to summarize the marginal dependence of injury frequency on each independent variable. These were of the form

$$Y = b_0 + b_i X_i \quad (i = 1,2,...7) \qquad (2)$$

The sample sizes varied for each of these cases since data for each variable were not available in all instances. Only two of the fitted regressions explained a significant (approximately 5% level) amount of the observed variation in injury frequency. The resultant regression equation for tons per worker was

$$Y = 225.5 + 0.09X_1 \qquad (3)$$

with a correlation coefficient of 0.27. The equation for container size was

$$Y = 125.7 + 3.7X_5 \qquad (4)$$

with a correlation coefficient of 0.39.

Although these relationships are not highly significant, there are definite indications that injury rates may increase with greater total tonnage handled, or be reduced by restricting the size of refuse containers. Because of the observed correlation of the two variables mentioned earlier, it is difficult to determine whether one is more important than the other. One would believe, *a priori*, that total tonnage handled should influence the total injury frequency rate while container size would influence the specific incidence of back injuries, strains and sprains, and lacerations. These injuries, incidentally, account for approximately 60 percent of the total injuries to refuse collection workers, according to the survey data.

Additional insight into the relationships discussed, or their elimination from consideration, is totally dependent on obtaining more accurate and valid data.

Individual Worker Analysis. Two responding cities — one with a collection force of 23 and the other with 60 men — provided detail data of injuries, time lost, and type of injury by individuals for nine and five years, respectively. The data are displayed in Table 4. Columns 2 to 10 show number of injuries and total number of days lost for the year. Column 11 provides the total number of injuries, total number of disabling injuries and total days lost for the period of employment. Column 12 (f) gives a simple injury frequency rate, while column 13 (F) provides the disabling injury frequency rate. Column 14 (S) shows the severity rate. The probable conservative nature of these frequency rates compared to Bureau of Labor Statistics rates was previously explained.

Table 5 provides an injury analysis for these cities. During the year 1964, 48 percent of the employees of City I were injured at least once. For City II, 73 percent were injured. It would seem extremely unlikely that this many 'accident prone' individuals could be hired by two widely separated cities.

TABLE 4

PERSONAL INJURY DATA

Col. 1	Col. 2		Col. 3		Col. 4		Col. 5		Col. 6		Col. 7		Col. 8		Col. 9		Col. 10		Col. 11			Col. 12	Col. 13	Col. 14
	1957		1958		1959		1960		1961		1962		1963		1964		1965		*Totals, 1957-1965*			injury freq rate f	Disabling injury freq rate F	Severity rate S
City I	NI[a]	TL[b]	NI[a]	TL[b]	NI[a]	TL[b]	NI[a]	TL[b]	NI[a]	TL[b]	NI[a]	TL[b]	NI[a]	TL[b]	NI[a]	TL[b]	NI[a]	TL[b]	NI[a]	DI[c]	TL[b]			
Worker A	1	6	1	0	0	0	0	0	2	0	0	0	1	6	2	30	2	23	9	6	65	500	333	3,610
Worker B	2	2	1	45	1	70	X												4	3	117	667	500	19,500
Worker C	1	6	0	0	0	0	1	132	2	18	X								4	3	156	400	300	15,600
Worker D	1	4	0	0	1	0	1	18	0	0	0	0	0	0	0	0	1	0	4	2	22	222	111	1,220
Worker E	–	–	2	26	1	58	3	160	0	0	1	0	1	0	X				8	4	244	668	334	20,300
Worker F	–	–	1	91	0	0	1	5	0	0	0	0	1	0	1	0	X		4	2	96	286	143	6,850
Worker G	–	–	1	45	1	7	1	1	0	0	1	28	X						4	4	81	400	400	8,100
Worker H	–	–	–	–	–	–	–	–	1	14	1	19	X						2	2	19	500	500	4,750
Worker I	–	–	–	–	–	–	–	–	2	77	0	0	0	0	2	14	1	0	5	3	91	500	300	9,100
Worker J	–	–	–	–	–	–	–	–	–	–	–	–	1	0	2	12	X		3	2	12	750	500	3,000
Worker K	–	–	–	–	–	–	–	–	–	–	–	–	–	–	2	77	1	5	3	3	82	750	750	20,500
City II																								
Worker A	–	–	–	–	–	–	–	–	1	5	2	19	X						3	3	24	750	750	4,920
Worker B	–	–	–	–	–	–	–	–	1	10	1	25	0	0	1	0	X		3	2	35	360	240	4,400
Worker C	–	–	–	–	–	–	–	–	2	8	0	0	0	0	2	24	X		4	3	32	480	360	4,020
Worker D	–	–	–	–	–	–	–	–	1	49	2	1	1	7	X				4	3	57	667	500	9,500
Worker E	–	–	–	–	–	–	–	–	1	18	1	9	2	11	2	17	X		6	4	55	720	480	6,910
Worker F	–	–	–	–	–	–	–	–	1	14	0	0	1	10	X				2	2	24	334	334	4,000
Worker G	–	–	–	–	–	–	–	–	1	11	2	47	0	0	1	5	X		4	4	63	480	480	7,920
Worker H	–	–	–	–	–	–	–	–	1	10	2	20	0	0	1	6	X		4	4	36	480	480	4,530
Worker I	–	–	–	–	–	–	–	–	1	10	1	8	2	29	X				4	4	47	667	667	7,850
Worker J	–	–	–	–	–	–	–	–	1	23	2	9	X						3	3	32	750	750	6,570
Worker K	–	–	–	–	–	–	–	–	1	40	0	0	0	0	1	62	X		2	2	102	240	240	12,800
Worker L	–	–	–	–	–	–	–	–	1	8	2	20	0	0	1	2	X		4	4	30	480	480	3,780
Worker M	–	–	–	–	–	–	–	–	1	12	1	0	2	1	X				4	2	13	667	334	2,330
Worker N	–	–	–	–	–	–	–	–	1	12	2	38	2	8	X				5	3	58	835	500	9,680
Worker O	–	–	–	–	–	–	–	–	–	–	2	22	3	28	X				5	5	50	1,250	1,250	12,500

[a] NI = Number of injuries

[b] TL = Time loss, days.

[c] DI = Number of disabling injuries.

NOTE: X in a column means no further record on the individual question.

Illness Data. Organizations responding to the questionnaire submitted little information pertaining to illness. Only one municipality indicated type of illness and number of occurrences. Nine municipalities indicated only the amount of sick leave time. The percent of sick leave noted for the above gives no indication of being in excess of the normal amount expected for all industries. The average was 2.82 percent, with a minimum of 1.67 percent and a maximum of 4.25 percent. The city reporting types of illness indicated that 40 percent of their sanitary workers' illnesses were confined to flu or upper respiratory infections; there was a 22 percent incidence of gastritis. The remaining 38 percent was scattered among various ailments.

TABLE 5

INJURY ANALYSIS FOR 1964

Item	City I	City II
Number of employees	23	60
Number of employees injured	11 (48%)	44 (73%)
Once	6 (26%)	29 (48%)
Twice	5 (22%)	10 (17%)
Three times		5 (8%)
Total number of injuries	16	64
Frequency (f)	348	532
Total number of lost time injuries	12	36
Frequency (F)	252	300
Total lost time	216	334
Severity (S)	4,700	2,780

CORPORATION WORKERS

Questionnaires were transmitted to 325 of America's largest corporations. The response was limited to 69, or 21 percent of those contacted. One organization submitted worthwhile and extensive information. The remaining 68 responses ranged from those which contracted for refuse disposal and gave no data, to those which indicated that no problem exists.

Discussion

The provision of adequate safety measures and controls for industrial employees, of all categories, requires a basic understanding of what problems actually exist. From the comments and lack of data received, the inevitable conclusion is that medical directors, safety supervisors, industrial hygienists, etc. of large corporations are not directing attention to the potentially hazardous nature of solid waste collection and disposal. The single exception was the reply of a safety supervisor for one large company, which segregated and tabulated occupational disability among workers handling solid wastes in that company. In spite of commendable attention to this group of employees, the company was unable to reduce its accident frequency rates to less than three times the rates published for miners (a high rate group) by the Bureau of Labor Statistics.

With this as an indication of the extent of the hazards involved in industrial sanitation work, the comments of other large companies may be revealing:

"We have no record of injuries or illness resulting to anyone from disposal of solid wastes" (an oil company).

"None of us can recall any injury or illness from solid waste disposal."

"This isn't a large problem in our corporation" (a metal products company).

"We have never experienced any illness or any disability accident attributable to this operation" (a meat packing company).

The majority of companies merely indicated their lack of any data concerning injury or illness to their sanitation workers.

In summary, the survey revealed that, among organizations maintaining surveillance on sanitation workers' occupational injuries, the average accident frequency rate of 156 was 4¼ times that of the highest rate (36.71) of any major industry (coal mining) reported by the National Safety Council for 1965 *(National Safety Council News,* September 1966).

This finding would seem to contradict the statement previously quoted from the American Public Works Association to the effect that refuse collection was not a hazardous occupation.[273]

Conclusions and Recommendations

There is strong evidence that solid waste handling is a hazardous occupation, both intrinsically and because insufficient attention has been paid to prevention of injury among sanitation workers. Definitive information is largely lacking. There is reason to believe that some of the high rates referred to in investigations reported in the literature, and implied in the survey just discussd, are due in considerable degree to the absence of safety programs. For example, in this survey, only 53 percent of those municipalities providing sufficient data for analysis conducted some kind of safety program.

It is admitted that the statistical analysis of injuries in this report is highly suspect: The response to the questionnaire was quite low, the questionnaire itself is suspect, and the data provided are of questionable validity in most instances. Nevertheless, it is not improbable that the data received represent a significant portion of the total available material. It is therefore submitted that the results — particularly the accident frequency rates — are sufficient to suggest a comprehensive investigation of sanitation safety to include not only municipal, but also industrial personnel, engaged in handling solid wastes. Even without further investigation, the institution of safety programs by municipal waste collection agencies and others would seem to promise some reduction in injury.[447, 449, 451] Driving safety programs for sanitation workers also may prove promising.[450]

However, a high-quality safety program encompassing all sanitation work — vehicle or equipment operation, manual labor and the like — should effect significant prevention of disability. For these reasons, the following action is recommended:

Alert safety agencies to the high accident frequency rates

Increase the amount of safety education directed at agencies and industries handling solid wastes

Institute plans for obtaining reliable data on all aspects of occupational injury and illness in this work

Institute further studies on the nature of the hazards and means for their prevention in sanitation work.

F. disaster

To obtain an estimate of the maximum effects of disaster on spread of disease through solid wastes, three studies on the relation of nuclear attack to disease were reviewed.[739, 740, 741]

In the shelters, periods of confinement to enteric diseases were estimated to be second only to respiratory diseases in incidence.[740, pp. 18, 22] This incidence was related to crowding and lack of facilities for good personal hygiene — especially for handwashing (pp. 18, 23, 27). In reference 740 (p. 27) the following statement appears: "There will be a very wide occurrence of more or less mild enteric disease within the shelter with the way being paved for the dissemination during the postattack period of more serious enteric disease such as typhoid fever and amebiasis." The possibility of the development of plague is noted in that work (p. 27).[740]

Reference 740 (p. B-3) also refers to the lack of information permitting calculation of acute as opposed to chronic disease prevalence. However, it was estimated that 94 percent of the total shelter population would be exposed to some communicable disease. Among the enteric diseases, greatest concern was expressed for the spread of shigellosis (p. 23); some cases of amebiasis and viral hepatitis also could be expected to occur. In those (probably) rare cases in which a typhoid carrier entered a shelter, the spread of infection could lead to a serious post-shelter risk of dissemination of typhoid fever, and thus could become a major problem in the population (p. 24).

It is stated in reference 740 (p. 27) that "arthropod-borne disease will not be a problem within the shelter, but the lack of facilities for good personal hygiene will lead to widespread louse and flea infestation which may well lead to the later development of epidemic typhus fever and plague in some parts of the country."

Of 51 diseases studied, 14 were considered to be related to environmental sanitations that would be significantly influenced by nuclear attack and the related waste disposal, pest, and vector conditions in the post-attack period.[739, p. i] Among the enteric infections, shigellosis, infectious hepatitis, salmonellosis, typhoid fever, and amebiasis were considered of special concern at such a time. The threat of mosquito-borne encephalitis was considered to be of equal concern, at least on a selective seasonal basis and in those parts of the nation where encephalitis is endemic. The difference in conditions conducive to the spread of other environmental diseases, such as plague, malaria, murine typhus, and the like were noted. It was stated that modification of the postattack environment as it would be related to the transmission of disease could be accomplished by institution of practices that have proven effective in the control of such diseases during peacetime. These sanitary countermeasures were noted to include refuse sanitation, arthropod control, rodent control, and rabies control (p. j).

In reference 740 (p. 2) the following statement appears: "Post-attack conditions may be favorable for a rapid increase in insect and rodent populations as a result of disruption of sanitary services and creation of extensive breeding and harborage areas. Survivors may be exposed to endemic diseases capable of rapid development in an uncontrolled environment. The surviving population (including sanitation workers) may be confined to shelters . . . for many days during which time fecal material and other organic wastes may accumulate in the shelter or in the area adjacent thereto, resulting in hazard of exposure to the 'filth' diseases. . . . Flies, mosquitoes, and other disease vectors may multiply rapidly as a result of cessation of controls and the creation of environmental conditions favorable to their growth."

This study considers that enteric diseases are the most important ones in the post-attack situation (p. 3). The providing of potable water for drinking and personal and household hygiene, safe disposal of body and solid organic waste, the providing of foods under sanitary conditions, and fly abatement measures were considered to be the most important factors in prevention of disease (p. 10). Quantitative factors in the production of solid wastes under various post-attack conditions were discussed (p. 41 ff).[740]

The relatively high level of sanitation in peacetime in the United States has resulted in a population which is lacking immunity to enteric diseases. The importance of this lack of protection is discussed with regard to breakdown of sanitary control — especially in the disposition of solid organic wastes, including feces (p. 64). Rodent-borne diseases are considered to be of relatively little importance in nuclear attack conditions. A similar conclusion is drawn with regard to mosquito-borne disease, with the possible exception of viral encephalitis (pp. 68–76).[740]

In summary, this study reported: "The fly-borne enteric diseases . . . may become a hazard in the postattack environment and will be limited only by the effectiveness of measures for the handling for human feces and the control of adult flies in the early post-attack. Also important in the early postattack environment is the control of mosquitoes (encephalitides) and fleas and ticks (leptospirosis, typhus) in areas experiencing epidemic or endemic outbreaks at time of attack" (p. 81).[740]

In another study,[741] crowding and bad sanitation following nuclear attack was thought to be a potential source of enteric and rodent-borne disease. The breakdown of public health controls was thought to represent a hazard in regard to malaria and other epidemic diseases (p. v-3, 4).

Two random quotations having implications for disaster situations may be of interest. In Eastern Europe, "the growing number of cases of Salmonella food poisoning may be explained by the expansion of communal feeding."[324 (abstract, p. 7)]

In Russia, the German occupation created conditions for the mass reproduction of "mouse-type rodents" associated with a considerable number of cases of tularemia.[379] With liberation of the area and resumption of normal household life, a sharp drop in the morbidity of tularemia occurred. The author feels that the mice were involved in the spread of tularemia.

Discussion

As previously stated in this report, the contribution of solid waste to disease cannot be considered alone: solid organic waste disposal and sanitary water supplies for drinking and personal hygiene share equal importance in the prevention of a number of diseases.

In spite of the general quality of the study[755] on waste disposal under conditions of nuclear warfare, the conclusions expressed seem somewhat optimistic in regard to the dissemination of disease under these conditions. In regard to civil disaster of limited or far less severity, however, this report is encouraging, unless return to normal conditions of sanitation were interfered with by prolonged strikes or civil strife.

In disaster situations, the setting up of food kitchens in a stricken area may help create problems of sanitation among which improper disposal of refuse could add to the total threat of disease.

Conclusions

Too little is known of solid waste/disease relationships to permit dependable prediction of their incidence and seriousness under emergency conditions involving prolonged disruption of services for the disposal of solid wastes. The studies reviewed seem not to have taken into account other host and environmental factors potentially modifying susceptibility among the victims of disaster.

Since the methods now used in this country to minimize the effects of local emergencies were not investigated, the degree to which they take into account the problems of solid waste was not determined.

Where recovery would be rapid and effective medical care readily available, however, the spread of disease from all sources would appear to be containable in areas of limited disaster.

Recommendations

British and German data on solid waste/disease relationships under wartime bombing conditions should be obtained and summarized, if available. The British have been relatively meticulous in their public health records, and may have such information.

Another source of information which should be summarized is that of the American and International Red Cross activities in disaster areas.

It would also be advisable to assemble all studies made in regard to Civil Defense, or on behalf of the Department of Defense on disaster sanitation.

Since something approaching the lack of sanitation in limited disaster can occur in uncontrolled urban expansion among low-income groups and migratory laborers, and in developing slum areas, greater information on the endemicity and epidemicity of disease in such areas, and the status of solid waste and other sanitary conditions, is needed.

An additional source of information could result from the sending of public health teams capable of studying the spread of disease in areas where disaster has disrupted normal sanitation. One of the missions of these teams would be to define both the problem of solid-waste disposal and the contribution of solid wastes in such situations to the dissemination of disease in the disaster populations.

Expanded reporting of diseases possibly related to solid waste should add to knowledge of their transmission under both non-emergency and emergency conditions.

As the total information on epidemiology and medical ecology is expanded, it may be possible to construct mathematical models or analogues from which the relative role of solid waste sanitation can be estimated in the majority of situations, including disaster. Coordination of epidemiologic and solid-waste research programming should be instituted to encourage the development of such models.

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APPENDIX A: RESEARCH recommendations

TABLE A-1 is a summary list of research topics discussed in succeeding pages of this appendix. Some are quite specific and deal with problems which can be clearly defined. Others are nebulous or sufficiently complex that no easy solution can be visualized. In any case, it is an imposing list because of the wide range shown. It may be best to consider the various research topics as 'a shopping list' for scientists and engineers who are interested in the field of solid wastes and public health. It would seem that almost everyone could find a research area which might benefit from his attention.

The list may also be considered as comprising a set of recommendations for future research and development work on recognized, real-life problems facing the country today. As such, the various research topics can serve as a guide for formulating and funding specific projects. A deficiency in the list is created by the fact that there was not time available to develop criteria necessary to permit assigning a priority to each topic.

A discussion of the material on which the list is based follows Table A-1

TABLE A-1

RESEARCH TOPICS (REPRESENTATIVE)

Systems Aspects

- System analysis of solid waste management, including disease
- Solid waste management criteria
- Formulation of a clear objective of solid waste–disease control measures
- Criteria and methods for measuring success of a system
- Consequences of failure of parts or all of a solid waste management system
- Requirements analysis for monitoring system
- New analytic procedures for identification and measurement of components, including microflora (monitoring)
- Methods for rapid screening of solid waste components for toxicity and disease potential
- Information storage and retrieval system for solid wastes
- Mathematical model of urban–rural interface emphasizing diseases and solid wastes

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Chemicals (see section on diseases associated with chemical wastes: Recommendations)

- Chemical factors in solid waste leading to disease (epidemiologic studies to identify hazards)
- Toxicology of components of solid waste
- Study of leaching and migration of chemical species through soil
- Study of chemical contamination of soil by specific problem compounds (e.g., heavy metals, nitrates)
- Study of waste contamination of air

(continued)

TABLE A-1 *(continued)*

Chemicals

Ways of using land disposal of wastes without affecting ground water quality (e.g., in landfills or gas-off)

Methods for tracing sources of chemical pollution

Microbiological Disease Agents and Disease Processes

Survivability and growth of mycotic disease agents in conjunction with sites of avian manure disposal; evaluation of potential hazards of using such areas for parks, etc.; migration of bacteria and viruses through soil from leached solid wastes

Methods for more rapid detection of pathogens

Survival and transmission of pathogens in solid wastes; treatment methods and factors effecting destruction

Occupational hazards study in sanitation and agricultural workers from waste handling

Role of solid waste in transmission of viruses into ground water

Effects of various treatment methods on virus removal from wastes

Mechanism of sludge–virus complex formation

Investigation into the bacteriocidal nature of seawater and survival of pathogens in seawater

Study on dispersal of solid wastes in the ocean and their possible relation to disease in marine organisms and in man

Interruption of cycles of zoonotic diseases, animal-to-animal and animal-to-man

Epidemiology of diseases having potential association with solid wastes

Vector control

Ecology of waste disposal sites in relation to fauna, flora, and disease agents

Waste Management

New concepts for isolating solid wastes from the environment

Solid waste management problems of small communities

Solid waste treatment system for communities of less than 10,000

New concepts for garbage disposal

Research into improved methods of treatment of specialized wastes

New uses for sludges and specialized wastes

Individual household treatment units

Education

See Recommendations under the various sections in disease

Safety

Disease spectrum of sanitation workers in comparison with general population

Accident causes among sanitation workers

Waste-handling hazards among other than sanitation personnel (agriculture, injury, households)

general considerations

THIS PORTION OF THE REPORT is concerned with a review of existing and past research activities relating solid wastes to public health problems, and with recommendations and conclusions for future research efforts. It is hoped that the organization of the material has permitted a logical and meaningful presentation of the more important areas needing attention. The level of effort devoted to this section did not permit a *uniform* breakdown to specific needed research projects; some of the work proposed for future includes additional surveys, literature reviews, and analyses to identify other specific research tasks.

Gilbertson[749] cites the fact that solid waste disposal has long been a neglected area of research as evidenced by the small amount of funds made available to workers in the field. Review of the literature makes this apparent — especially in the field of solid waste/public health interface. Since solid waste management is such an extraordinarily complex problem, systematic technical and socioeconomic approaches will be required to gain and maintain control of the undertaking. To date, no adequate inputs have been developed for a definitive systems analysis, and much research will be required before progress can be made in this area. Not only are health hazards difficult to establish in many cases, but goals have been stated only in general terms. These goals must be converted into waste management criteria and related to control of health hazards. In this respect, it is interesting to note[237] that the need for criteria has been recognized in a recommendation for the development of an integrated program of waste management in a new community.

Some general statements about research in solid waste/public health relationships deserve mention. The public health problems are more severe in the smaller communities than in large urban areas. There is thus a real need for a complete study of environmental sanitation on a national scale.[562] Furthermore, a great deal of information is in the literature. Considerable information is available about how to solve many of the problems. What is required is research on how to apply what is already known.[561]

An inescapable conclusion reached in the literature review is that some form of systematic information gathering activity in solid waste technology should be continued; perhaps an annual annotated bibliography, up-dating previous years, would be in order. This activity should be broadened to include aspects not encompassed in the current project.

systems analysis

SYSTEMS ANALYSIS has been used extensively by government agencies and industry in choosing among alternative approaches to various complex problems. In general, the techniques are used to deal with problems in which it is desirable to minimize or maximize an objective which is subject to requirements or constraints. The logic of systems analysis allows an ordering of the total problem, the incorporation of nontechnical factors, and the presentation of alternatives to the decision-makers in clear and unambiguous terms. A clear lay presentation of the subject of systems analysis, and how it may be applied to complex problems, has been published.[713]

The recognized complexity of the overall waste management problem, now, and in years to come, led to consideration of the use of systems analysis techniques in developing solutions to the waste management problem in the State of California. In an initial study[736] it was concluded that perhaps the *only* successful approach to waste management would be through the avenue of systems analysis. Lynn[647] discussed the use of systems analysis in solid waste problems. He pointed out that solutions based on systems analysis provided sound bases for reaching decisions.

Supporting the contention that systems analysis has an important role to play in solid waste management, is a project being supported jointly by the State of California Department of Public Health, and the Solid Wastes Program of the U.S. Public Health Service. This study (contract NO. 15100), being conducted by Aerojet-General Corporation, involves the systems approach to the solution of the solid waste problems in Fresno, California, and is considered a demonstration program.

The lack of analytical data makes it clear that there is a need for research to obtain data to be used as input for systems analysis. One result of the systems analysis approach is the identification of fruitful areas of research and development.

Certain important aspects of systems analysis stand out, about which comment should be made. These relate to system evaluation and offer several important areas of needed research. System evaluation has been stated to consist of four steps:[564]

1) Formulation of the objective

2) Identification of the proper criteria to be used in measuring success

3) Determination and explanation of the degree of success

4) Recommendations for further program activity.

Each step is, of course, extremely important, and much could be written about each one and the need for investigations. Concerning the first step, it is necessary to decide, in terms of public health, the goal to be reached according to some time frame. This is a complex problem; insofar as the available literature is concerned, it has not been adequately considered from a systems viewpoint.

The identification of the proper criteria is, if anything, more complex than the first — mainly because of the myriad of constraints which exist. These constraints also have an influence on a practical objective. Costs, public interest, political, social and economic considerations, technological limitations — these and other factors all influence criteria, as well as the objective.

The third step offers fairly clean-cut areas for specific research tasks in identifying products (organisms or chemicals) which are undesirable, and in developing techniques for quantitatively determining them. The possibility of developing a rapid screening method for potentially hazardous waste materials has been suggested.[218] Other suggestions include a systematic monitoring system for pesticides to detect incipient problems,[264, 385] a method for evaluating the performance of an incinerator,[285] new analytical procedures for measuring industrial wastes,[267] and methods for detecting and measuring microflora.[33] The foregoing is not an exhaustive list, but demonstrates the type of research recommended in the literature.

The last step mentioned above would receive attention after operational systems were available.

Very little other evidence of direct application of systems analysis to waste management was found in the literature surveyed. There were found, however, indications that more organizations are considering the value of a systems approach, and of developing some of the data required as input for such analyses.[749] The need for mathematical models has been recognized in the context of a systems analysis approach to urban planning.[234] The advantages of an optimized system are also discussed. The stated need for planned management of natural resources as a total system[615] implies the use of the techniques being discussed. Engineering parametric studies are also desirable.[145]

It is believed that the main contribution of the systems analysis approach to solid waste–public health relationships may be in the development of criteria and in identifying interface problem areas, such as is being done in the Fresno project mentioned earlier. Furthermore, such analysis should be one concerned with the total solid waste management problem of which one part is that concerned with public health. Thus, it is recommended that in a systems analysis program dealing with solid waste management, full recognition of the public health aspects be included.

disease agents and solid wastes

DISEASE AGENTS, as used here, fall into two categories: chemical agents and biological agents. There is a continuing need in both categories to catalog specific agents and components of solid wastes which may offer potential hazards because of the changing nature of solid wastes over a period of years.

Chemical Agents

Research on chemical wastes has been concerned mainly with identifying etiologic agents, factors involved in dispersion of agents, and prevention of dispersion. It is through chemicals that the greatest interfaces exist between solid wastes and pollution of soil, water, and air. Various investigations have demonstrated specific problems which may be directly related to solid wastes. They are important to the present discussion because they point out certain research needs.

Thus, infant methemoglobinemia has been cited as being caused by polluted water (wells) contaminated from solid wastes.[277, 353] A need for further study to permit verification and development of comprehensive preventive measures is required, as are investigations of other factors influencing the occurrence of the disease.[353]

There have been several investigations concerned with the importance of leaching in landfills in polluting groundwaters. It has been stated that if a landfill does not intercept ground water, no impairment of water supplies will occur.[473] Pollution will occur in the immediate vicinity if there is contact. Apparently, leached inorganic materials pose more of a problem than organic wastes because they tend to migrate farther[416, 576] Ground water pollution sources are difficult to trace and may ruin an aquifer for years, or permanently.[430] This potential indicates a real need to examine solid waste disposal techniques and sites to assure that leaching will not produce public health problems. There are a number of investigations dealing with the topic of effects of solid waste disposal on water quality, but these efforts do not deal with specific disease or public health problems.[10, 105, 559, 576, 636] It is also in this area of concern that differences between liquid and solid wastes begin to disappear. The problem is one of water pollution — no matter what the source. Once the pollutant is in the water, it may never be known whether it entered the water originally as liquid or as solid waste.

Land areas suitable for sanitary landfill are becoming more and more difficult to find. Research has been recommended to find ways to use abandoned gravel pits and underground cavities without endangering the quality of ground water.[211, 478, 636]

The types of information needed in the future to evaluate specific problems of land disposal and public health problems relate to a number of different disciplines. There is need for epidemiologic studies to identify public health problems.[478, 634] Identification of potential hazardous waste components has already been mentioned. Ways of rendering chemicals innocuous should be investigated, or easily degradable materials developed.[209, 247, 257] Chemical reactions during migration in the soil may change the chemical species of concern[385, 416]; thus there is a need to investigate the ultimate fate and nature of materials leached from solid wastes. Factors influencing leaching and movement of compounds through soil need also to be studied.[559] An important area of concern is the determination of acceptable or tolerable levels of the myriad of materials which may escape into the environment[316, 629]; new materials, about which little or nothing is known, are being introduced at a rapid rate and many contribute to solid wastes.[628]

Two types of chemicals are of special concern: carcinogens and pesticides. Carcinogens, arising in water supplies contaminated either from liquid or solid wastes, have received considerable attention.[44, 45, 46, 61, 132, 144, 316, 387, 704] Specifically, the literature points out the requirement for studies of movement of carcinogenic materials through soils,[44, 45] because of the conflicting nature of the data and lack of full definition of the problem.

Aromatic hydrocarbons have been shown to be leached from macadam roads, and also to occur in sewage sludge when treatment is incomplete.[3] Hueper points out that most proofs of carcinogenesis in humans are limited to occupational exposures but that there is most probably a general population exposure of unknown magnitude.[387] Other reports[51, 67, 79] substantiate this claim in one way or another and give emphasis to the urgent need for comprehensive chemical, experimental, and epidemiologic studies to determine actual hazards.

Pesticides may find their way into solid wastes by three principal routes: containers containing pesticides,[15] in food wastes,[385] and by direct application to wastes to control pests.[209] Once in the wastes, they are subjected to leaching as are other compounds. Although there is little or no medical evidence that adverse effects have occurred, there is cause for concern.[102, 264, 323, 395] The Secretary of the Interior has been quoted as stating, "Not until we have a systematic monitoring scheme, designed to tell us how much of what is where, will we be able to detect incipient problems early enough to prevent damage."[264] Additional investigations are needed to determine safe limits of lifetime exposures to toxic materials, the nature of injuries if excessive amounts are absorbed, means for detecting subclinical effects, and therapeutic measures.[385] This particular reference is concerned primarily with pesticides in foods, but most of the discussion is equally applicable to solid wastes. Possible chemical changes in pesticide residues are also of interest, and there is a definite requirement for better analytical methods and schemes for detection and identification.

In a discussion concerning water quality and goiter,[14] additional studies are recommended on the nature of chemical pollutants and their physiological significance. By implication, the role of solid wastes should be included in such studies. From other reports[85, 132, 577, 578] although they do not deal with solid wastes specifically, requirements can be derived from investigations of the role of solid wastes in other disease or nutritional problems involving trace elements.

Insofar as air pollution by solid wastes is concerned, the main routes are via burning or bacterial activity within a disposal site. Specific respiratory disease occurrence has been cited as due to burning in dumps,[4, 347] but the evidence is circumstantial — pointing up the need for more exhaustive investigations. The desirability of an impervious barrier in landfills to prevent escape of obnoxious gases has been discussed.[445] Few other specific solid wastes–atmospheric-borne disease investigations have apparently been done, though air pollution by burning is widely recognized.

Biological Agents

Disease is not a result of simple contact between host and disease agent. Health and disease are results of complex interplay among many factors. Frequently, some or many of the factors are unknown. In many reports, the connection between a waste treatment process and disease is not mentioned; yet, implicit in selection of the process is the knowledge (or hope) that "proper" treatment will decrease the number and incidence of disease organisms. Thus, much of the surveyed literature dealing with waste is only indirectly concerned with disease. On the other hand, the medical literature deals in great detail with specific diseases, their diagnosis, causative agents, intermediate hosts, and therapy, but is rarely concerned with any solid waste aspects.

Of primary concern (in regard to free biological agents of disease) is whether or not the method of disposal, or the products of treatment which are to be used in agriculture or other industries, permit survival and transmission of disease organisms to humans, plants, or animals.[480]

An important factor at disposal sites is the possibility that organisms may migrate through the soil as a result of leaching and movement of ground water, and pollute water supplies. This process appears to be a more important problem for chemical contaminants (see the next section) than for organisms because of natural mortality and/or removal of viruses and cells by filtering processes. However, as early as 1927 *E. coli* was known to remain viable for 31 months in experimentally polluted ground water.[43] Later studies[325, 478] provided data concerning movement of bacteria through soils and factors affecting it. There have been a few investigations which indicate that detergents affect movement of bacteria through soil.[257] This topic is one which should receive additional attention in research.

Viruses are said to survive better in treated water than in polluted water.[12] This finding has been confirmed by Clarke *et al.* who found that viruses survive longer in relatively unpolluted waters,[16] whereas bacterial survival is directly related to pollution levels. This finding might prove to be the basis for an imaginative new approach to waste treatment.

The sludge from various types of sewage treatment presents a solid waste problem of considerable magnitude.[216] Sludge may be used directly in agriculture or in a process such as composting. In agricultural use of sludges, viable organisms may be carried to water supplies by surface runoff[629, 634]; the pathogens may offer an occupational exposure to agriculture or sanitation workers,[11, 211, 324, 480] or may contaminate foodstuffs.[480] Because of these potential problems, the study of pathogen survival and methods for assuring their destruction become important.

Various studies[16, 21, 33, 39, 56-I, 80, 215, 216, 480, 491, 494] indicate that the activated sludge process removes a high percentage of pathogens but that the sludge must receive additional treatment to be considered safe. Special sludges (e.g., sludge containing copper) may pose few problems as far as disease organisms are concerned[215]; however, they may create a toxic hazard. This report and other sources[480, 491] indicate that only heat-dried sludge may be considered free of biological disease agents.

Research is needed in methods of economically dehydrating sludge, so that its fertilizer potential can be realized.[494] Additional information on removal of pathogens by various treatment processes is still required.[21, 39] Better methods for detection and enumeration of pathogens are also needed.[33] Other processes such as anaerobic biological treatment[249] and lime softening[19] have been investigated as removers of pathogens. It is apparent from the literature that there is still opportunity for advances through research in the removal or destruction of pathogens in sludges.

Farm manures are a source of transmission to man of some animal diseases[6, 324, 425]; one preventive suggestion found was to burn suspect manures.[425] There appears to be a need to evaluate further the magnitude of this problem and to develop simplified control measures. Avian manures pose a special problem in histoplasmosis transmission (see section on fungus disease). Histoplasmosis is considered a 'disease of nature', localized to certain regions of the country and spread to humans from a reservoir in the soil.[326] The occurrence of the disease organisms has been correlated with avian or bat droppings.[35, 89, 173, 175, 326, 502, 503, 504] However, there is a question as to whether or not the organism can be supported within the avian body[504]; it may be that soil enriched with manure provides an appropriate ecologic niche for the organisms.[503] Histoplasmosis has been found to persist in manure-enriched soil for as long as three years,[326] and there are various theories as to how it is disseminated. Since it is unlikely that the disease agent can be eradicated, the only feasible method of control is through vaccination.[502] A skin test is currently in use for epidemiologic study, but a more refined tool is needed for adequate surveys.[504]

The literature on histoplasmosis, and other mycoses as well, shows the need for additional research in certain areas related to solid wastes (for example, more thorough investigation of the environmental conditions required for survival, persistence and growth, and methods for eradication). The true role of avian and bat manure needs to be determined. Since, in landfill disposal methods, the reclaimed area may be later used for other purposes involving contact by humans, the persistence and movement of the fungi and subsequent hazards need also to be determined.

Composting solid wastes, either singly or in various combinations, has been or is being done at various sites. Pertinent to the present discussion, however, is the potential hazard that compost may present to the public health.

Golueke and Gotaas[622] point out that the temperature produced in proper composting exceeds the thermal death points for common pathogens and parasites, and that no public health problem then exists. Many references recognize that the heat produced in composting, when properly carried out, may be expected to kill pathogenic bacteria, viruses, fungi, protozoan cysts, and helminth eggs.[371, 406, 613, 642, 652] There appears to be an occupational hazard to compost workers in adding night soil or sewage sludge.[314] The same report indicates that improper composting produces a health hazard (flies) almost as at a garbage dump. Suitable microbiologic tests must be performed to demonstrate that a given process of composting does, in fact, destroy undesirable organisms.[602]

Various studies have been concerned with survival or destruction of pathogenic or parasitic organisms in composting, and in developing ways to assure their destruction. Thus, when Salmonella and Shigella organisms were not killed in a sawdust–sludge compost process, adding well-rotted barnyard manure resulted in higher temperatures which did destroy these organisms.[602] In windrow composting of refuse and sludge, on the other hand, additives had no effect.[504]

One of the advantages of composting is the avoidance of public health problems by disposing of special wastes. Thus, a disagreeable fly problem is avoided[626] by composting fruit processing wastes, as well as poultry and other animal manures.[170, 612]

There are a few claims of exceptional value of composting in improving livestock growth,[652] and in controlling nematodes which attack potatoes and tomatoes,[606] perhaps by unknown antibiotics produced in compost.

Extensive research in the field of composting, insofar as disease is concerned, does not appear to be needed. If the procedures are properly carried out, there appears to be little hazard. The problem seems to be primarily one of proper equipment design, training operators in the efficient running of the plant, and reliable monitoring of the process. Studies of occupational exposures might be desirable if composting plants become economical in the United States and more such plants are built. Some research on methods of detecting and identifying pathogens might be profitable, as in other areas of waste management.

Exceptional effects on growth of cattle through the feeding of compost materials are claimed. The evidence seems tenuous, but may warrant further investigation, particularly of the possibility that production of antibiotics is involved.

As an aside from disease, the problem in composting is to make it economical since this is the usual failing of the process. To this end, research to find uses for compost appears to be most needed — a development that would help greatly in the overall solid waste management problem.

Some consideration should be given to certain marine aspects of disposal of sludges. In using ocean disposal methods, it is important to dilute the wastes and dispose of them so that they do not return to shore. Research to gain an understanding of the action of waves, currents, and winds in the dispersion process is thus important.[149] In addition, there are a number of reports indicating that seawater apparently has bacteriocidal properties[126, 372, 853]; research in this area is required. In addition, *E. coli* may not be the indicator organism of choice in marine pollution studies because it does not survive well in seawater.[42] Fecal streptococci may prove superior, but proof is required.

There are animal reservoirs of disease agents which can attack man. Some of these animals (e.g., the rat) are associated with solid wastes. Similarly, there may exist, in solid wastes, disease organisms which may be carried by arthropods (especially flies) to human beings.

One way to reduce the disease hazard is to prevent vermin from utilizing solid wastes for either harborage or for nutrients (i.e., to control the environment). Landfill operations (with or without compaction or shredding), disposal into sewers after grinding and subsequent sewage treatment, incineration, composting, and ocean disposal are commonly used to keep refuse away from vermin. Any improvements in, or more widespread use of, these techniques may be expected to improve the public health. Among these might be listed better waste collection and transportation systems.

Fly control has been recognized as a major problem relating to refuse and public health,[75] thus, research in this area may be expected to assume a high priority. In considering controls from the solid waste point of view, there are several possible categories for research:

1) Totally new concepts for garbage collection and disposal or treatment
2) Better means of isolating garbage (e.g., better containers)
3) More adequate pesticide application wherever wastes are stored (e.g., some acceptable means of pesticide application or other treatment at the household level)
4) Development of a continuing program of education of the public so that adequate procedures will be followed
5) Improved methods of treatment of specialized wastes (e.g., manures, food-processing wastes).

Much research has been conducted on various facets of zoonoses (animal diseases transmissible to man). This includes such items as descriptions of life cycles, host–disease relationships, epidemiologic studies, and determinations of geographical limits of particular foci. In this work, however, very little reference is made to solid wastes. It has been noted that contact occurs between commensal rodents (which live in part on solid wastes) and wild (rural) rodents.[165, 172, 569, 570, 566] The cited reports deal with investigations on plague.

Stark and Miles[566] determined that when commensal and wild rat populations intermingled, there was an interchange of fleas — suggesting that the plague could thus be transmitted from a wild population to the commensal one, with subsequent increased hazard to man. In their research these authors worked with eleven species of fleas and nine species of mammals, further indicating the complexity of the problem. Kartman *et al.*[569] also demonstrated the transfer of fleas from wild to domestic rodents. It has been stated[172] that an outbreak of plague in Los Angeles in 1924 was due to a transfer of the disease from ground squirrels to domestic rats. Several reports[165, 172, 573, 566, 569] state or strongly imply the need to keep wild and commensal populations separated. The problem is well described by Meyer[165] who points out that during urbanization of an area, there is a period of joint tenancy by humans and by wild and commensal rodents — a situation theoretically conducive to the transmission of plague to humans.

Since the contribution of solid wastes to potential disease transmission is not de-

fined, research is needed to develop models of the urbanization phenomenon (which include the ecologic aspects of zoonoses and wastes) for later use in investigations of potential epidemic hazards. A number of suggestions for purely biological or medical investigations into zoonoses are beyond the scope of this report in that they may be only remotely (or not at all) related to the role of solid wastes in public health problems.

Although arthropods other than flies may act as carriers of disease organisms, flies by far overshadow the others. Because of this, the present discussion will be limited to flies, although it is possible that the same statements could apply to other arthropods (see sections on fly- and mosquito-borne disease).

Although flies are seldom true intermediate hosts in a disease cycle,[72] they are responsible for dispersal of pathogenic agents; they may be used as indicator organisms, reflecting the sanitation level of a community.[75, 86] The use of insecticides such as chlordane and DDT have proven only temporary measures for fly control since resistant strains of the insects arise.[75] The control of flies within a community requires such intimate intervention into the lives of residents that adequate methods for complete control have never been developed. Current practices (namely, education on how to handle garbage and on the importance of using tight containers for garbage) are unsatisfactory.[348] The use of garbage grinders — eliminating garbage storage and collection — appears to be the best present solution to the problem in residential areas.

Flies, being the problem they are, receive continuing attention in studies relating to specific problems[e.g., 76, 188, 301, 391, 544] and in more general treatments.[e.g., 75, 77] Special note should be taken of the use of radioisotope-labeled flies in a study of dispersal in a metropolitan area[187] — one of the first field applications of radioisotopes. Studies have been carried out on the persistence of disease organisms in various stages of fly development. Larvae grown in contaminated media have been found to retain viable disease organisms through various stages of metamorphosis.[184, 300]

Additional suggestions for research related to vectors are contained in the main sections of this report dealing with fly- and mosquito-borne disease.

APPENDIX B: SOLID WASTE SOURCES AND CONSTITUENTS

Municipal Refuse[231]

Source	*Waste*	*Composition*	*Means of treatment or disposal*
Households, restaurants, institutions, stores, markets[231]	Garbage[231]	Wastes from preparation, cooking and serving of food; market wastes from handling, storage and sale of food[231]	Grinding, incineration, landfill, composting, hog feeding[231]
	Rubbish[231]	Paper, cartons, boxes, barrels, wood, excelsior, tree branches, yard trimmings, wood furniture, bedding, dunnage, metals, tin cans, metal furniture, dirt, glass, crockery, minerals[231]	Salvage, incineration, landfill, composting, dumping[231]
	Ashes[a, 231]	Residue from fires[231]	Landfill, dumping[231]
Streets, sidewalks, alleys, vacant lots[231]	Street refuse[231]	Sweepings, dirt, leaves, catch basin dirt, contents of litter receptacles, bird excreta[231]	Incineration, landfill, dumping[231]
	Dead animals[231]	Cats, dogs, horses, cows, marine animals, etc.[231]	Incineration, rendering, explosive destruction[231]
	Abandoned vehicles[231]	Unwanted cars and trucks left on public property[231]	Salvage, dumping[231]
Factories, power plants[231]	Industrial wastes[b, 231]	Food processing wastes, boiler house cinders, lumber scraps, metal scraps, shavings, etc.[231]	Incineration, landfill, salvage[231]
Urban renewal, expressways, etc.[231]	Demolition wastes[231]	Lumber, pipes, brick masonry, asphaltic material and other construction materials from razed buildings and structures; bat guano, pigeon excreta[231]	Incineration, landfill, dumping, salvage[231]
New construction, remodeling[231]	Construction wastes[231]	Scrap lumber, pipe, concrete, other construction materials[231]	Incineration, landfill, dumping, salvage[231]
Households, hotels, hospitals, institutions, stores, industry[231]	Special wastes[231]	Hazardous solids and liquids, explosives, pathologic wastes, radioactive wastes[231]	Incineration, landfill, burial, salvage[231]
Sewage treatment plants, lagoons, septic tanks[231]	Sewage treatment residue[c, 231]	Solids from coarse screening and grit chambers, sludge[231]	Incineration, landfill, composting, fertilizing[231]

[a] See "Fly Ash" under Waste Treatment Products for Chemical Composition.[223]

[b] See "Industrial Wastes" for detail characteristics and composition.

[c] See "Sludge" under Waste Treatment Products for Chemical Composition.

AGRICULTURE REFUSE

Source	*Waste*	*Composition*	*Means of treatment or disposal*
Farms, ranches, livestock feeders, and growers[209]	Refuse	Same as Municipal Refuse	Same as Municipal Refuse
Farms, ranches, livestock feeders, and growers[209]	Crop residue[209]	Cornstalks, tree prunings, pea vines, sugarcane stalks (bagasse), green drop, cull fruit, cull vegetables, rice, barley, wheat and oats stubble, rice hulls. Fertilizer[e] and insecticide[e] residue[209]	Plowed back into the land, incineration, stock feed[209]
	Animal manure[248] (Paunch manure)[248]	Lignaceous and fibrous organic matter, nitrogen, phosphorus, potassium,[248] volatile acids, proteins, fats, carbohydrates[238]	Fertilizer, composting[248] Stock feed[238]
	Poultry manure[248]	Same as animal manure[248]	Fertilizer, composting, lagooning[248]

INDUSTRIAL WASTES

Food and kindred product industries[205]	Fruit, vegetable and citrus[215]		Hull, rinds, cores, seeds, vines, leaves, tops, roots, trimmings, pulps, peelings, hydrochloric acid[251] (used in processing)	Screening, lagooning, soil absorption, spray irrigation, reclamation[219, 215, p. 6]
Canning[205]	Cobs, shells, stalks, straws[205, 219]	High in suspended solids (liquid waste) colloidal and dissolved organic matter[246, 205]		
Vegetable oil refining			"Still pitch" — tarry residue, fatty acids, sodium hydroxide, trichlolethylene[219]	Reclamation[219]
Dairy[208, 251]	Dilutions of whole milk, separated milk, buttermilk and whey[251]	High in dissolved organic matter, mainly protein, fat, and lactose[251]	N, CaO, K_2O, P_2O_5, Fe, Cl, SiO_2[281]	Aeration, trickling filter, activated sludge[251]

[e] See "Industrial Wastes" for characteristics.

Industrial Wastes *(continued)*

Source	*Waste*	*Characteristics*	*Composition*	*Means of treatment or disposal*
Slaughtering of animals, rendering of bones and fats, residues in condensates, grease and wash water[251,282]	Manure, paunch manure, blood, flesh, fat particles, hair, bones, oil, grease[282]		N, NH_3, NH_2, NO_3, $NaCl$[282]	Reclamation, screening, trickling filters,[251] chlorination[246]
Breweries and distilleries[251,215]	Spent grain, spent hops, yeast, alkalis, amyl alcohol, dissolved organic solids containing nitrogen and fermented starches[215]	High in dissolved organic solids, containing nitrogen and fermented starches or their products[251]	Amyl alcohol[235] (from processing)	Recovery, centrifugation and evaporation, trickling filtration, stock feeds,[251] fertilizer[215]
Pharmaceutical[251]	Microorganisms, organic chemicals[251]	High in suspended and dissolved organic matter, including vitamins[251]	Aniline, phenols[235]	Evaporation, incineration, stock feeds[251]
Textile mill products[251]	Textiles, i.e., cotton, wool, and silk[246]	Highly alkaline, colored, high BOD and temperature, high suspended solids[251,246]	H_2SO_4,NaOH, aniline chlorine[235] Starch, malt, tin & iron salts, dyes, bleach, fibers, minerals[246]	Neutralization, precipitation, trickling filtration, aeration, recovery[219,251]
Cooking of fibers, desizing of fabrics[251]		Same as textile mill products	For complete list of chemicals used in textile industry, see reference[279]	
	Rayon, other man-made materials, i.e., Acrilan, Dynel, Orlon, Nylon, etc.[279]	Acidic, alkaline, inorganic[246]	Sulfides and polysulfides, colloidal sulfur, NaOH, H_2SO_4, $ZnSO_4$, HCl, $NaHSO_4$, H_2S, $CaSO_4$[246]; acrylonitrile, phenol, HNO_3[235]; ammonia, adiponitrile, hexamethylenediamine, sodium carbonate, alcohols, ketones[283]	Reclamation, neutralization trickling filtration, lagooning[246,219]

INDUSTRIAL WASTES *(continued)*

Source	*Waste*	*Characteristics*	*Composition*	*Means of treatment or disposal*
Laundry [246, 251]		High turbidity, and alkalinity [246, 251]	Spent soaps, synthetic detergents, bleaches, dirt and grease [215, 246]	Screening, precipitation, flotation, adsorption [251]
Lumber and wood products (forest, mills, factories) [251, 215, 219, 246]	Pulp and paper [251, 215]	High or low pH; colored; high suspended, colloidal, and dissolved solids; inorganic fillers [251]	Sawmill usage (sawdust, shavings, wood chips), wood flour [219]; soda, sulfate, sulfite [246]	Reclamation, incineration, soil conditioning [219]
	Organic, inorganic, toxic, suspended and dissolved solids of lignin, resins, soda, ash, fiber, adhesives, ink, fats, soaps, tallow [246]		Sodium lignate, sodium resinate, complex organosulfur compounds, some fiber in relatively dilute solutions, [256] sulfites, [225] mercaptans, sulfides, disulfides, sulfates, terpenes, carbohydrates, CaO, SO_2, N, PO_4 [246]	Reclamation, settling, lagooning, biological treatment, aeration [219, 251]
Chemical plants (general)		Toxic [246, 235]	Acrylonitrile, aniline,[f] amyl alcohol, carbon disulfide, carbon tetrachloride, chlorine, hydrogen cyanide, hydrochloric acid, phenol, sulfuric acid, toluene, xylene, dinitrobenzene, dimethyl sulfate, ethylene, chlorohydrin, benzene, metallic compounds of lead, arsenic and mercury [235]	Reclamation, lagooning and all other known methods of treatment [245]
	Fumes and/or dust [704]		Arsenic [704]	

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[f] Most common and troublesome toxics.

INDUSTRIAL WASTES *(continued)*

Source	*Waste*	*Characteristics*	*Composition*	*Means of treatment or disposal*
Chemical plants *(continued)*	Particulate clouds and dusts[221]		Mn, Va, Cd, Be, Fe, Zn, and their oxides[221]	
	Weed killer[430]		2-4-D[430]	Sewage[430]
	Cyanide waste[243]	Toxic to aquatic life[243]	Cyanides[243]	Ponding[243]
	Plastics, synthetic resins		Acrolein, acrylonitrile, formaldehyde, phenols, trichlorethylene[235]	Reclamation, incineration[219]
Aircraft manufacturing industry[478]	Cd and Cr^{+6}[478]	Traces of metals[478]	Cd and Cr^{+6}[478]	Leaching pits[478]
Waste treatment plants[402]	Well-digested sludge[402]	Blackish, amorphous, nonplastic material[402]	Mg, Ca, Zn, Cr, Sn, Mn, Fe, Cu, Pb[402]	Anaerobic decomposition of organic waste solids[402]
Petroleum industry[251]	Spent chemical[419]	Liquid wastes with oil, acid and alkaline solutions, inorganic salts, organic acids and phenols, etc.[419]	Clays, H_2SO_4, H_3PO_4[419]	Streams[419]
Drilling		Oil, brine, chemicals[246]	Sodium, calcium, magnesium, chlorine, SO_4, bromine[246]	Separation, evaporation, lagooning[246]
Storage	Muds, salt, oils, natural gas[251]			Separation, evaporation, lagooning[246]
Distillation[251]	Acid sludges, miscellaneous oils[251]	Insoluble organic and inorganic salts, sulfur compounds, sulfonic and napthenic acids, insoluble mercaptides, oil-water emulsions, soaps, waxy emulsions, oxides of metal, phenolic compounds[276, 251]	Na_2CO_3, $(NH_4)_2S$, Na_2S, sulfates, acid sulfates, H_2S, $NaOH$, NH_4OH, $Ca(OH)_2$, $(NH_4)_2SO_4$, NH_4Cl, phenols[251]	Settling, filtration reclamation, evaporation[246]

INDUSTRIAL WASTES *(continued)*

Source	*Waste*	*Characteristics*	*Composition*	*Means of treatment or disposal*
Treating[251]		See "Distillation"[276, 251]	See "Distillation"; also lead, copper, calcium[251]	Reclamation, settling filtration, evaporation, neutralization[246]
Recovery[251]		See "Distillation"; also organic esters[276,251]	See "Distillation"; also iron[251]	See "Treating"[246]
Leather and leather products[246, 251]	Tanneries[246]	Organic and inorganic, high BOD – lime sludge, hair, fleshing, tan liquor, bleach liquor, salt, blood, dirt, chrome[246]	Chromium, sulfuric acid, nitrogen, $CaCO_3$, D_2O_5, K_2O, Fe[280]	Sedimentation, lagooning[246]
Energy producing industry[223, 207, 240] Pulverized coal-fired plants; stoker-fired, cyclone-fired plants; and wet-bottom pulverized coal-fired plants	Fly-ash[223]	Hollow spheres of fused or partially fused silicate glass or as small solid spheres of fused silicates, iron oxides or silica, unburned carbon and mineral[223]	Silicates, iron oxide, silica[223]	Sold for use in concrete,[223] landfills, etc.
Electrical industry[298]	Ash[298]	Dust[298]	Silicates and alumninates of Fe, Cu, Mg with small percentages of Na, K[298]	
Metal finishing industry[215, 258, 242, 254, 435]	Pickling and washing liquors[215]	Toxic, waste waters[215]	Cu and Cu alloys[215]	Sewage[215]
	Acid wastes[254]	Harmful to aquatic life,[254] salts of metals[258, 242, 254, 435]	Cu, Ni, Zn, Cr, Fe[258, 242, 254, 235]	Sewage[258, 242, 254, 435]
Rubber and miscel. plastic products[251]	Rubber[251]	High BOD, odor, high suspended solids, variable pH, high chlorides[251]	Sulfuric acid, trichlorethylene, xylene, amyl alcohol, aniline benzene, chromium formaldehyde[235]	Aeration, chlorination, sulfonation, biological treatment[251]

INDUSTRIAL WASTES *(continued)*

Source	*Waste*	*Characteristics*	*Composition*	*Means of treatment or disposal*
Washing of latex; coagulated rubber; exuded impurities from crude rubber; rejects, cuttings, mold flashings, trims, excess extrusions [219, 251]	Scraps from molding, extrusion, rejects, trimming and finishing [219]			
Explosives [251] Washing TNT and guncotton for purification, washing and pickling of cartridges [251]		TNT, colored, acid, odorous, and contains organic acids and alcohol from powder and cotton, metals, acid, oils and soaps [251]	H_2SO_4, HNO_3, NO_2SO_3, picric acid, TNT isomers, copper, zinc, nitrogen, toluene [246]	Dilution, neutralization, lagooning, flotation, precipitation, aeration, chlorination [251]
Phosphate and phosphorous [251]	Washing, screening, floating rock, condenser bleed-off [251]	Clays, slimes, tallows, low pH, high suspended solids [251]	Phosphorous, silica, fluoride	Settling, clarification (mechanical), lagooning [251]
Fertilizers			Nitrogen, phosphorous, potassium, sulfuric acid, traces of other chemicals	
Coke by-products	Slag from ovens, ammonia still waste, spent acids and phenols	Suspended solids, volatile suspended solids, organic and NH_3–N, phenol, cyanide, acids, alkalis [251]	Ammonia, benzene, H_2SO_4, phenol [276, 251]	Discharged to sewers, dumped, incineration [276]
Industrial, not otherwise identified [416, 293, 200, 279]	Inorganic industrial waste or stabilization [416]	Metals and compounds thereof [416]	Na, K, Ca, chlorides, sulfates, bicarbonates, nitrates, phosphates, fluorides, borates, chromates, etc. [416]	
	Metallic fumes and dusts [293]		Pb, Va, As, Be and compounds thereof [293]	
	Industrial wastes [200]	Mineral fines [200]	Chromates, heavy metals [200]	Underground aquifers [200]

INDUSTRIAL WASTES *(continued)*

Source	*Waste*	*Characteristics*	*Composition*	*Means of treatment or disposal*
	Laboratory wastes[479]		Metallic ions, phenolics, cyanides, oils, synthetic fibers, pharmaceuticals, rubber chemicals[479]	Landfill or dump[479]
	Industrial wastes	Toxic metals[235]	Pb, Be[235]	
Insecticides	Washing and purification of products[251]	High organic matter, toxic, acidic[251] Chlorinated hydrocarbons: toxaphene, benzene, hexachloride, DDT, aldrin, endrin, dieldrin, lindane, chlordane, methoxychlor, heptachlor[323]	Carbon, hydrogen, chlorine, carbon disulfide, carbon tetrachloride	See Chemical plants (general)
		Organic phosphorous compounds: parathion, Malathion, phosdrin, tetraethyl, pyrophosphate[323]	Phosphorous, oxygen, carbon, hydrogen, carbon disulfide, carbon, tetrachloride[323]	
		Other organic compounds[323]	Carbonates, dinitrophenols, organic sulfur compounds, organic mercurials, rotenone, pyrethrum, nicotine, strychnine[323]	
		Inorganic substances[323]	Copper sulfate, arsenate of lead, compounds of chlorine and fluorine, zinc phosphide, thallium sulfate, sodium fluoracetate[323]	

APPENDIX C: DESCRIPTOR GLOSSARY

primary search descriptors

FIRST ORDER

Refuse
Waste

SECOND ORDER

Ashes
Cinders
Compost
Dirt
Dust
Garbage
Junk
Litter
Manures
Garbage
Sewage
Sludge
Trash

secondary search descriptors

FIRST ORDER

Disease Terminology

Bacteria
Bacteriology
Disease
Fungi
Helminths
Microbiology
Moulds
Parasites
Pathogens
Pathology
Protozoa
Rickettsia
Viruses

Disposal Terminology

Collection
Disposal
Dump(s)
Landfill(s)
Processing
Sites (Disposal, Treatment, etc.)
Treatment

Host Terminology

Animals (Other)
Host(s)
Rats
Rodents

Vector Terminology

Arthropod(s)
Entomology (Medical)
Fleas
Flies (Fly)
Lice (Louse)
Mosquitoes
Tick(s)
Vector
Vermin

Safety Terminology

Accident
Frequency Rate
Hazard(s)
Injury
Safety
Sanitation Men
Severity Rate
Workers (Sanitation, Sewage Treatment Plant, etc.)

General Health Terminology

Epidemiology
Environmental Health
Health
Hygiene
Preventive Medicine
Public Health
Sanitation, Sanitary
Sanitary Engineering

Chemical Terms

Carcinogens
Chemical(s)
Insecticides
Intoxicants
Pesticides
Poison(s)
Toxicology
Toxins

Miscellaneous Items

Chlorination
Components
Constituents
Contamination
Decomposition
Demolition
Dump(s)
Fertilizer
Harbor(age)
Humus
Incinerator(s)
Irrigation
Lagoon(s)
Landfill(s)
Leaching
Pond(s)
Reclamation
Soil
Utilization (use)
Wrecking

SECOND ORDER

Second-order descriptors in this category are included in the body of the glossary which follows.

DESCRIPTOR GLOSSARY

ABATEMENT
ABATTOIRS
ABATTOIR WORKERS
ABORTED FETUS
ABSORPTION
ACAROLOGY, MEDICAL
ACCELERATED SLUDGE DIGESTION
ACCEPTABLE DAILY INTAKE
ACCIDENTS
ACETALDEHYDE
ACETIC ACID
ACHROMOBACTER SPECIES
ACID (ITY)
ACID CALCIUM SULFITE LIQUORS
ACID MINE DRAINAGE
ACID-FAST BACILLI
ACIDIFICATION
ACRYLONITRILE
ACTINOMYCES
ACTINOMYCETES
ACTINOMYCOSIS
ACTIVATED
ACUTE
ADAPTATION
ADDITIVES
ADENOVIRUS
ADMISSION
ADMIXTURES
ADRENAL CORTEX
ADSORBENTS
ADVANCED WASTE TREATMENT
AEDES AEGYPTI
AEDES ALBOPICTUS
AEDES SOLLICITANS
AEDES VEXANS
AERATION
AEROBES (BIC)
AEROSOLS
AESTHETIC
AGE
AGE GROUP
AGENCY (AGENCIES)
AGENTS
AGGREGATE (LIGHTWEIGHT)
AGAUTI
AGRANULOCYTOSIS
AGRICULTURE (AL)
AIRBORNE
AIR POLUTION
AIR STRIPPING
AL, SEE ALUMINUM
ALCES AMERICANA
ALCOHOL (S)
ALDRIN
ALKALI (S)
ALKALIGENES
ALKALINE (ITY)
ALKALOIDS
ALKYL BENZENE SULFONATES (ABS)
ALOPEX LAGOPUS
ALTITUDE
ALUMINA
ALUMINUM
AMEBAS
AMEBIASIS
AMERICAN CUTANEOUS AND MUCOCUTANEOUS LEISHMANIASIS
AMINO ACID (S)
AMINOAZO DYES
AMINOTRIAZOLE
AMMONIA
AMPHIZENOSES
ANAEROBIC
ANAEROBIOSIS
ANALYSIS
ANALYTICAL PROCEDURES
ANALYTICAL TECHNIQUES, SEE ANALYTICAL PROCEDURES
ANCHOCERCA
ANCYLOSTOMA CANIUM
ANCYLOSETOMA BRAZILIENSE
ANCYLOSTOMA CEYLANICAM
ANCYLOSTOMA DUOBENALE
ANEMIAS
ANGIOSTRONGYLUS CANTONEUSIS
ANHYDROUS HCE
ANIMAL
ANOZEMIA
ANOPHELES
ANOPHELES STEPHENSI
ANTHRACITE
ANTHRAPOZOONOSES
ANTHRAX
ANTHRAX BACILLI
ANTHROPONOSES
ANTIBODIES
ANTIBIOTIC INHIBITORS
ANTIGEN
ANTIGENIC COMPONENTS
ANTIGENICITY
ANTIMONY
ANTISEPTIC
ANTS
API SEPARATORS
APLASTIC ANEMIA
APODEMUS FLAVICOLLIS
APOPHALLUS
AQUEOUS COMBUSTION
AQUIFERS
ARAMITE
ARBOVIRUSES
ARCTIC
AREA
ARGON
ARID AREA
AROMATIC AMINES
AROMATIC RING COMPOUNDS
ARSENIC
ARSENIC TRIOXIDE
ARSENICAL EPITHELIOMA
ARSENICAL INTOXICATION
ARSENIOUS ACID
ARSENIOUS ANHYDRIDE
ARTESIAN WELLS
ARTHROPOD (S)
ARTHROSPORES
ARTIFICIAL FEEDING STUFFS
ARTIFICIAL TOPSOIL
ASBESTOS
ASCARIS EGGS
ASCARIASIS
ASPECTS
ASPERIGILLUS NIGER
ASPERIGILLUS SYDOWII
ASPHYXIATION
ASTHMA
ATMOSPHERIC CONTAMINANTS
ATROPINE
ATTACK RATE
AUREOBASIDIUM PULLULANS
AUSTRALIS GROUP
AUTOMOBILE EXHAUST
AVIAN SPECIES
AVIANS
A.W.T.

BABOON
BABY
BACILLI
BACILLUS COLI COMMUNIS
BACILLI DYSENTERIAE (SHIGA)
BACILLI PARATYPHOSUS B. SCHOTTMULLER
BACTERIA
BACTERIA, MESOPHILIC
BACTERIA, PATHOGENIC
BACTERIA, SAPROPHYTIC
BACTERIA, THERMOPHILIC
BACTERIOLOGY (ICAL)
BACTERIOLOPHAGE (S)
BAGASSE
BAGGAGE
BAKERS
BALANCE
BALANTIDIOSIS
BALANTIDIUM COLI
BALING
BANDICOTA BENGALENSIS
BANGALARE PROCESS
BARNACLES
BAT
BDELLONYSSUS BACOTI
BEARS
BED BUGS
BEDDING
BEEF
BEETLES
BEHAVIOR
BENZENE
BENZOL
BENZANTHRACENE, 1, 2, 9, 10 - DIMETHYL
BENZPYRENE, 3, 4
BERYLLIUM
BHC
BIOCHEMICAL DEGRADATION
BIOCHEMICAL DISTURBANCE
BOD
BOD REMOVAL
BIOLOGIC INERTNESS
BIOLOGIC LAW
BIOLOGICAL
BIONOMICS
BIOTIC
BIRD
BISON
BITES (BITING)
BITUMINOIS COAL
BLACK ASH
BLACK LIQUOR
BLASTOMYCOSIS
BLEACH LIQUORS
BLEACHES (ING)
BLOCKING
BLOOD
BLOOD DYSCRASIAS
BLOOD_SUCKING ARTHROPODS
BLOW FLY
BLUEBOTTLE (FLY)
BONE MEAL
BORATES
BORIC ACID
B. SEE BORON
BORON

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BORRELIA
BORRELIA BUTTONI
BOTULISM
BOVINE ANIMALS
BREAST FEEDING
BREEDING
BRINES
BRONCHIAL EPITHELIAL HYPERPLASIA
BRONCHITIS
BRUCELLOSIS
BUBONIC PLAGUE
BUFFALO
BUILDING MATERIALS
[illegible]
BURNING
BURRS
BURYING (BURIAL, BURIED)
BUSINESS
BUTCHER SHOPS
BUTCHERS
BUTYL ALCOHOL
BY-PRODUCTS

CA, SEE CALCIUM
CEC, PREPARED ANTIGEN
CABLE SYSTEMS
CADAVER PROCESSING PLANTS
CAKE
CALCIFICATION
CALCIUM
CALCIUM OVIDE
CALF (CALVES)
CALIFORNIA
CALLIPHORIDS
CALLITROGA MACELLARIA
CAMP ACTION
CANCER
CANEFIELDS
CANE SUGAR
CANIS LATRANS
CANIS LUPUS
CAPACITY
CAPILLARY
CARBOHYDRATES
CARBON
CARBON DIOXIDE
CARBON DISULFIDE
CARBON FILTER TECHNIQUE
CARBON MONOXIDE
CARBON SLURRY
CARBON TETRACHLORIDE
CARBONATE
CARCINOGENIC AROMATIC HYDROCARBONS
CARCINOGENIC EFFECT
CARCINOGENS
CARCINOMA OF THE BRONCHIAL GLANDS
CARCINOMA OF THE PLEURA
CARCINOMA OF THE RIBS
CARDIOVASCULAR DISEASES
CARIBOU
CARNIVOROUS MAMMALS
CARRIER
CASE RATIO
CAT
CATALYTIC
CATERERS
CATTLE
CAUSTIC
CAVES
CENTRAL EUROPEAN ENCEPHALITIS
CENTRAL EUROPEAN TICK-BORNE ENCEPHALITIS
CERAMIC PRODUCTS
CENTRALIZATION
CENTRIFUGES (AL, ING)
CERATOPHYLLUS ACUTUS
CERATOHPYLLUS FASCIATUS
CERATOPHYLLUS SILVANTIERI
CERATOPHYLLUS SPECIES
CERNUS CANADENSIS
CESIUM
CESSPOOLS
CESTODES
CHAIN
CHEESE
CHELATION
CHEMICAL (S)
CHEMICAL INDUSTRY
COD
CHEMOPROPHYLAXIS
CHEMOTHERAPEUTIC AGENTS
CHICKEN
CHICKEN HOUSES
CHIGGERS
CHILD (CHILDREN)
CHILOMASTIX
CHIPMUNK (S)
CHLORAMINE
CHLORAMPHENICOL
CHLORDANE
CHLORIDES
CHLORINATED
CHLORINATED HYDROCARBON (S)
CHLORINE (ATION)
CHLORTHION
CHOLANTHRENE, 20- METHYL
CHOLERA
CHOLERA INFANTUM
CHOLESTEROL
CHROMATES
CHROMIC ACID
CHROMIUM
CHRONIC
CHRYSOMYIA BEZZIANA
CILIATE
CITELLUS LATERALIS TESCORUM
CITRUS CROP
CITY DUMPS
CIVIL ENGINEERING CONCEPTS
CLARIFIER
CLEANLINESS
CLETHRIONOMYS GAPPERI
CLIMATE
CLINKER
CLOGGING
CLONORCHIS SINENSIS
CLOSTRIDIA
CLOSTRIDIUM PERFRINGENS
CLOSTRIDIUM WELCHII
CLOTHING
CMC
C:N RATIO (CARBON-NITROGEN RATIO)
CO, SEE CARBON MONOXIDE
CO_2, SEE CARBON DIOXIDE
COAGULATION
COAL
COALFIELD REGION
COASTAL WATERS
COAST DEER
COBALT
COBALT AREAS
COBALT ARSENIDE
COCCIDIODES IMMITIS
COCCIDIOIDOMYCOSIS
COCHLIOMYIA MACELLARIA
COCKROACHES, BROWN BANDED
COCKROACHES, GERMAN
COCKROACHES, ORIENTAL
COEXISTENCE
COFFEE
COLIFORM
COLIFORM INDEX
COLIFORM ORGANISMS
COLITIS
COLLAGEN FORMATION
COLLECTION
COLLOIDAL SYSTEM
COLLOIDS
COLONIES
COLORADO TICK FEVER
COMBUSTIBLE
COMBUSTION
COMFORT
COMMENSAL (ISM)
COMMERCIAL POISONS
COMMINUTE (COMMINUTORS, ING)
COMMUNAL FEEDING
COMMUNICABLE DISEASE CONTROL
COMMUNITY (TIES, SEE METROPOLITAN AREA)
COMPACTING
COMPETITIVE BIDDING
COMPLEMENT FIXATION
COMPLEX LANGAT NEGISHI
COMPOSITION
COMPOSE (ING)
COMPRESSION
COMPUTER
CONCENTRATION
CONCRETE
CONCURRENT ILLNESS
CONDEMNED MEAT
CONDENSATE
CONDITION (ING)
CONDUCTIVE
CONFECTIONERS
CONFINEMENT
CONJUNCTIONAL FLUID
CONJUNCTIONAL SURFACE
CONJUNCTIVITIS
CONTACT
CONTAINERS
CONSTITUENTS
CONSTRAINTS
CONSTRUCTION
CONSTRUCTION WORK
CONSUMER
CONTACT AERATION
CONTAINER
CONTAMINANT (S, ATION)
CONTROL
CONVALESCENT CARRIER
CONVERTING
CONVEYANCE
CONVEYANCE TO THE OCEAN
COOKS (ING)
COPPER
COPPER CYANIDE
COPROHAGONS BEETLES
COSTLY
COST (S), SEE ECONOMICS
COTTON-TAIL RABBIT
COUGAR
COUNT
COUNTRY
COWSHEDS
COXSACKIE VIRUS

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COYOTE (S)
CRAGS
CRANE OPERATOR
REATININE
CRESOLS
CRICKETS
CROP
CROWDING
CRYOGENIC SCRUBBING
CRYPTOCOCCOSIS
CRYPTOCOTYLE
CRYSTALLIZATION
CTENOCEPHALIDES CANIS
CTENOCEPHOLUS CANIS
CTENOPSYLLUS MUSCULI
CTENOPSYLLUS SEGNIS
CTF VIRUS
CULEX ANNULIROSTRIS
CULEX FATIGANS
CULEX NIGRIPOLPUS
CULEX SALINARIUS
CULEX TARSALIS
CULICONE MOSQUITOES
CULISITA MALANURA
CUIM
CULTIVATION
CULTURAL SOPHISTICATION
CUTANEOUS CREEPING ERUPTION
CUTANEOUS HOOKWORM INFECTION
CUTANEOUS, INNOCULATION
CUTANEOUS LEISHMANIASIS
CUTANEOUS LARVA MIGRANS
CYANIDES
CYCLONES
CYCTICERCUS CELLULOSAL
CYSTS

DAIRY COMMUNITY
DAIRY
DANO DIGESTER
DANO PLANT
DDT
DDVP
DEAD
DEATHS
DEBRIS
DECANTED
DECOMPOSITION
DEEP FREEZE STORAGE
DEEP-WELL INJECTION
DEHYDRATED (TION)
DELIVERY
DEMETON
DENGUE

DENGUE COMPLEX
DENSITY
DENTAL EPIDEMIOLOGY
D.O.
DERMACENTAR ANDERSONI
DERMACENTOR VARIABILIS
DERMATITIS HERPETIFORMS
DESERTED BUILDINGS
DETECTION
DETERGENTS
DEVELOPMENTAL STAGES
DEWATERED (ING)
DEXTRIN
DIAGNOSIS
DIALYSIS
DIAPERS
DIAPHRAGM
DIARRHEA
DIARRHEA, WHITE
DIARRHEAL DISEASE
DIAZINON
DIBENZANTHRACENE, 1,2,5,6,
DICROCOELIUM DENDRITICUM
DICHROMATE VALUE
DIELDRIN
DIFFERENTIAL DIAGNOSIS
DIGESTER (S, TION)
DIISOPROPYL FLUOROPHOSPHATE
DIMETHYLAMINE
DIMETHYL NITROSAMINE
DILUTION
DIPHTHERIA
DIPHYLLOBOTHRIUM LATUM
DIPYLIDIUM CANIUM
DIRECT COMBUSTION DEVICE
DIRT
DISC
DISEASE
DISEASE, FOOT AND MOUTH
DISINFECTION
DISPERSION (DISPERSAL)
DISPOSAL
DISPOSAL WELL
DISSEMINATION
DISSOLVED SOLIDS
DISTILLATION
DISTILLERS SLOPS
DISTILLERY
DOG
DOMESTIC, (RESIDENTIAL, HOUSEHOLD)
DOMESTIC CARNIVORES
DONORS
DOSAGE DELIVERY
DOSE (DOSAGE)

DOUGLAS SQUIRREL
DRACUNCULUS HOSPES
DRACUNCULUS MEDINENSIS
DRAINAGE
DRINKING
DROPPINGS
DROSOPHILA
DRUGS
DRY SEASON
DRYING
DUCKS
DUMP (ED)
DUNG
DUODENITIS
DUST
DYSENTERY
DYSENTERY, AMOEBIC
DYSENTERY, BACILLARY
DYSPEPSIA

EAR INFECTIONS
EARTHWORMS
EASTERN ENCEPHALITIS
EATING
ECHINOCOCCUS
ECHINOCOCCUS GRANULOSUS
ECHINOCOCCUS MULTILOCULARIS
ECHO VIRUS
ECOLOGY (ECOLOGICAL, ECOLOGIC)
ECOLOGY, LAWS OF
ECONOMICS
ECOSYSTEM
ECTOPARASITES
EDIBLE OIL
EDUCATION
EELWORM
EFFECT (S)
EFFICACY
EFFLUENT (S)
EGG BREAKING
EGG POWDER
EGG PRODUCTS
EGG SALAD
EGGS
ELECTRIC DUSTER
ELECTRICAL COALESCENCE
ELECTRICAL DISCHARGE
ELECTRICAL FORCES
ELECTRICIANS
ELECTROCHEMISTRY
ELECTRODIALYSIS
ELECTROENCEPHALOGRAMS
ELECTROLYTIC

ELECTROSTATIC
ELEMENTS
ELIMINATING
ELK
ELUTRIATION
EMUSIONS
ENCAPSULATION
ENCEPHALITIS
ENDAMOEBA HISTOLYTICA
ENDEMIC
ENDEMIC AREA
ENDEMIC CHAIN
ENDEMIC FOCI
ENDEMIC TYPHUS
ENDEMICITY STUDIES
ENDOBRONCHIOLAR
ENDOGENOUS OXIDATION
ENDOGENOUS RESPIRATION
ENDRIN
ENRICHMENT
ENSILAGE
ENTAMOEBA HARTMANNI
ENTERCOLITIS
ENTERIC FEVER
ENTERITIS
ENTEROBIUS VERMICULARIS
ENTEROCOCCI
ENTEROPATHOGENIC BACTERIA
ENTEROPATHOGENIC ESCHERICHIA COLI
ENTEROPATHOGENS
ENTEROVIRUSES
ENTOMOLOGY
ENTRAINED AIR
ENVIRONMENT (AL)
ENZOOTIC
ENZYME ACTION
EOSINOPHILIC MENINGOENCEPHALITIS
EPIDEMICS
EPIDEMIOLOGICAL
EPIZOOTIC
EQUINE ENCEPHALITIS
(EASTERN & WESTERN)
EQUINE ENCEPHALOMYELITIS
EQUIPMENT
EQUIPMENT USE
ERADICATION
ERETHIZON DORSATUM (PORCUPINES)
ERIPYSELOID
ERISTALIS TENAX
ERYPYSELOTHRIX INSIDIOSA
ERYSIPELAS
ESCHERICHIA COLI
ESHINOSTOMES
ESTABLISHMENT, SEE AESTHETIC

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ETHER
ETHIONINE
ETHNIC ORIGIN
EUROPEAN TICK
EUTECTIC
EVAPORATION
EVIDENCE
EXAMINATION
EXCREMENT
EVOLUTION
EXCRETA
EXPERIMENTS
EXPLOSION (S)
EXPOSURE
EXTRACTION
EYE

F, SEE FLUORINE
FABRIC COLLECTORS
FACILITY
FACTORS
FAECAL MATTER (SEE FECAL MATERIAL)
FAECAL POLLUTION (SEE FECES, POLLUTION)
FAMILY (IES)
FAMILY IXODIDAE
FANNIA CANICULARIS
FANNIA DUSIO
FARM (ING)
FASCIALOPSIS
FASCIOLA HEPATICA
FAT (S)
FATALITY RATE (SEE DEATH RATE)
FATERA INDICA
FATIGUE
FAUNA
FE, SEE IRON
FEATHERS
FECAL MATERIAL
FECAL STREPTOCOCCI
FECES
FECES TRANSMITTING INSECTS
FELDSPAR
FELIS CONCOLOR
FERAL MAMMALS
FERROUS OXIDE
FERTILIZER
FIBROUS MATERIALS
FIELD SPREADING
FILARIASIS
FILLER
FILTER (S)
FILTH
FILTRATION
FILTRATION RESISTANCE
FIRST-AID ROOM
FISH
FISH, (TROUT)
FISHER (FISHERMEN)
FLAKE
FLAME DRYING SYSTEM
FLEA
FLEA POOL
FLEA, RAT
FLEAS AND FLEA SPECIES
FLESH
FLESHINGS
FLIES, SEE FLY
FLOATING POLYMER
FLOCCULATION
FLOORS
FLOTATION
FLOW
FLUKE
FLUORIDE
FLUORINE
FLUSH TOILETS
FLY (FLIES, MUSCA DOMESTICA)
FLY ASH
FLY-MICROBE ASSOCIATION
FOAM (FOAMING)
FOCUS (FOCI)
FODDER
FOOD
FOOD-BORNE INFECTION
FOOD POISONING
FOOT AND MOUTH DISEASE
FORESTS
FORMALDEHYDE
FORMIC ACID
FOWL
FOX (FOXES)
FRACTIONATION
FREEZING
FRESHWATER
FRIJOES
FRONT END LOADING SYSTEM
FROTHING
FUME
FUNAMBULUS
FUNABULUS PENENTII
FUNGUS (FUNGI)

GALLUNDA ELLIOTE
GAME HANDLERS
GANGRENE
GARBAGE
GAS (ES)
GASOLINE
GASTRITIS
GASTRODISCOIDES
GASTROENTERITIS
GASTRO-INTESTINAL DISEASE
GASTRO-INTESTINAL INFECTIONS
GEOGRAPHIC DIFFERENCES
GEOGRAPHIC LOCALIZATION
GEOLOGY
GEOTRICHUM CANDIDUM
GERBIL
GIARDIA INTESTINALIS
GIARDIA LAMBIA
GIARDIASIS
GLANDERS
GLASS
GLUCOSE
GNATHOSTOMA
GNATHOSTOMA HISPIDIUM
GNATHOSTOMA NEOPLASTICUM
GNATHOSTOMA SPINIGERUM
GOATS
GOB
GOITER
GOITROGENIC SUBSTANCES
GONGYLONEMA
GOUT
GRAIN
GRAM-NEGATIVE
GRANARIES
GRASS LAND TREATMENT
GRAVITATIONAL FORCE
GRAVITY SEPARATORS
GRAY FOX
GREASE
GREEN DROP
GRID COUNTS
GRINDERS (GRINDING)
GRIT DEPOSITS
GROUNDWATER
GROUP (S)
GROUP BARBOVIRUSES
GROWTH
GUINEA PIGS
GUINEA WORM
GUMS

HABIT
HABITATION
HAEMAG-GLUTININ-INHIBITION
HAIRS
HAMMERMILL SYSTEM
HAMSTERS
HANDLERS
HANSEN BACILLI
HARDNESS
HAZARDOUS OPERATION
HAZARDS
HEALTH
HEAT
HELMINTH DISEASE
HELMINTHOLOGY
HELMINTHS
HEMICELLULOSES
HEMOLYTIC ENTEROCOCCI
HEMOLYTIC STREPTOCOCCI
HEPATITIS
HEPTACHLOR
HERBICIDES
HERBIVAROUS MAMMALS
HERD
HEREDITARY
HERMETIA ILLUCENS
HETEROPHYIDAE
HEXANE
HEXAVALENT CHROMIUM
HIGH-RATE
HIPPELATES PUSIO
HIPPURIC ACID
HISTOPLASMA
HISTOPLASMA CAPSULATUM
HISTOPLASMIN TESTS
HISTOPLASMOSIS
HISTORY
HOG CHOLERA
HOG HAIR
HOGS, SEE SWINE
HOLISM
HOME ACCIDENTS
HOMESTEADERS
HOOKWORM
HOOVES
HOPLOPSYLLUS ANOMALUS
HORMAT PROCESS
HORNS
HORSEFLIES
HORSES
HORTICULTURE (AL)
HOSPITAL
HOST (S)
HOST CONTAMINANT RELATIONSHIP
HOUSEFLY
HOUSEHOLD
HOUSE (HOUSING)
HUMAN

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HUMITDITY
HUMUS
HUNTERS
HYDATIDOSIS
HYDRATION
HYDRAULIC STRUCTURES
HYDROCARBONS
HYDROCHLORIC ACID
HYDROFLUORIC
ACID
HYDROGEN SULFIDE
HYDROSTATIC PRESSURE
HYGIENIC
HYMENOLEPIS NANA
HYPERENDEMIC SENSITIVITY
HYPERKERATOSES
HYPERSENSITIVITY
HYPOCHLORITE PROCESS
HYSTRICHOPSYLLA LINSDALEI

ICE
IMMUNE SERA
IMMUNITY
IMMUNIZATION
IMPURITY
INACTIVE DUSTS
INCIDENCE
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UNTREATED
URBAN AREAS
URBANIZATION
UREA
URETHAN
URIC ACID
URINE
UTENSILES
UTILIZED (UTILIZING,
    UTILIZATION)

VACUUM
VALUE (SEE ECONOMICS)
VAN MAANEN SYSTEM
VAPORIZATION
VAPOR PRESSURE
VASCULAR DISORDER
VECTOR POTENTIAL
VECTORS
VEGETABLE
VEGETATION
VEHICLES
VERMIN
VERTEBRATES
VETERINARIANS
VESICULAR EXANTHEMIA
VIBRATION
VIBRIO
VIBRIO CHOLERAE
VIBRIO FETUS
VIBRIOSIS
VILLAGE
VINEYARDS
VINYL ACETATE
VIRAL
VIRULENCE
VIRUS (ES)
VIRUS TITER
VISCERA
VISCERAL LEISHMANIASIS
VITAMIN
VOLATILE
VOMIT
VUEPES FUERA

WALRUS
WAPITI
WAR (WARFARE)
WAR-TIME
WAREHOUSE
WASH SINK
WASTE
WASTES, BEAMHOUSE
WASTES, BREWERY
WASTES, CANNERY
WASTES, CHROMIUM-BEARING
WASTES, CREAMERY
WASTES, DEMOLITION & CONSTRUCTION
WASTES, ELECTROPLATING
WASTES, FAT REFINING
WASTES, FOOD INDUSTRY
WASTES, FOOD PROCESSING
WASTES, GAS & COKE PLATE
WASTES, HYDROGEN CHLORIDE
WATER
WATER-BORNE
WATER-CLOSETS
WATERCOURSE
WATERCRESS
WATER-FRONT ZONE
WATER-QUALITY STANDARDS
WATER RESOURCES MANAGEMENT
WATERSHED MANAGEMENT
WAVE ACTION
WAX (ES)
WEANLING DIARRHEA
WEED(S)
WEED SEEDS
WEIL'S DISEASE
WELLS
WESTERN EQUINE ENCEPHALOMYELITIS
WESTERN HEMISPHERE
WET
WHALES
WHIPWORM
WHITE ARSENIC
WHITE-TAILED DEER
WILD
WILDCAT
WILDERNESS RECREATIONAL AREAS
WIND ACTION
WINDOW SCREENS
WINDROW
WINE RESIDUES
WINTER DIARRHEA
WOLF (WOLVES)
WOOD
WOODCUTTERS
WOODLANDS
WOOL
WOOL GREASE
WOOL WASTES
WOOLSORTER'S DISEASE
WORK (WORKER, WORKING)
WORM
WOUNDS
WUCHERERIA BANCROFTI
WUCHERERIA (BRUGIA) MALAYI

XENOPYSYLLA ASTIA
XENOPSYLLA CHEOPIS
XYLENE
X-RAYS

YAWS
YEAST(S)
YELLOW FEVER
YELLOW FEVER VIRUS
YIELDS
YOUNG CHILDREN

ZINC
ZONE(S) (ZONING)
ZONE OF INFECTION
ZOOANTHROPONOSES
ZOONOSES
ZOOPLANKTON

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